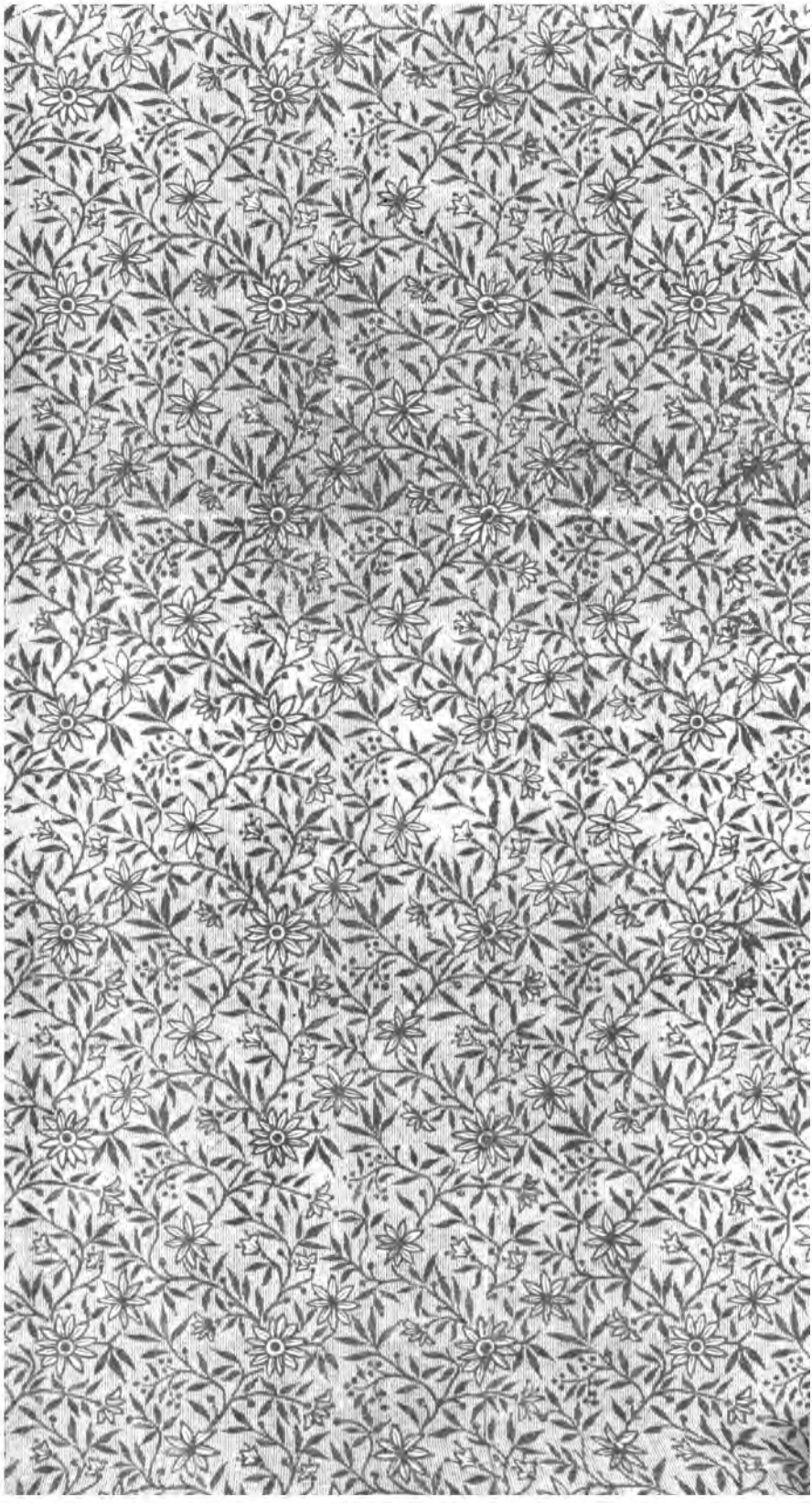

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ESSAYS

ON

HYSTERIA, BRAIN-TUMOR

AND

SOME OTHER CASES OF NERVOUS DISEASE

WITH THE

COMPLIMENTS OF THE AUTHOR

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CONTENTS.

HYSTERIA AND OTHER NEUROLOGICAL PAPERS.

I.

SOME CONSIDERATIONS ON HYSTERIA.[1]

NOTWITHSTANDING the voluminous literature which exists on hysteria, something always remains to observe and describe in it. And this is to be expected when it is remembered that hysteria implies disarrangement of the functions of any part of the nervous system — in its four spheres of intelligence, mobility, sensibility, and visceral neurility. Every advance in our knowledge of these mysterious functions must, therefore, lead to some new point of view in regard to hysteria, or to mental, motor, sensory, or visceral neurosis. Is it possible at the present day to formulate the fundamental condition of hysteria in such a way as to bring it into harmony with the facts of the hysterical temperament, of the general neurotic diathesis, of the vaso-motor spasms, of the special, mental, motor, and sensory phenomena of hysteria, and of the relations of the developed disease to the reproductive organs on the one hand, and to moral and social conditions on the other?

It seems to me that we can assert the following to

[1] The nucleus of this paper was read before the Neurological Section of the Academy of Medicine, June 11, 1886.

be the twofold condition fundamental to hysteria: There is in it a congenital or acquired deficiency in the power of nerve-elements to effect the storage of force in nerve-tissues.

This can only be overcome by increasing the amount of stimulus to which these elements are subjected. Conversely, the elements of those centres, which are subjected to a preponderance of stimulus, will perform the function of storage most effectively, and, in so doing, will acquire preponderance over the others. And this is done by the sensory centres of the brain.

These centres, connected with the nerves of special sense and of common sensibility, are, from the beginning of life, exposed to the most incessant stimulation, fom the constant impact upon them of centripetal impressions. The registration of these impressions is attended by chemical synthesis within the cells (Meynert) by "negative work" (Wundt). Such synthesis implies the storage of oxygen into complex chemical compounds, in which it becomes latent. These may be called force-compounds, because upon their explosive decomposition depends the liberation of energy, or force, the "positive work."[1]

Thus the synthetic nutritive processes of the central nervous tissues are closely associated with their functional stimulation through the arrival of impressions from the periphery. "The optic nerve, which resembles in structure the central white substance, undergoes changes within two or three days of extra-uterine life, which far exceed those changes which would take

[1] The negative work is the first result of centripetal stimulation of a nerve-centre (central galvanization). During it no tangible phenomenon occurs. It is followed by the positive work, centrifugal impulse resulting in muscular contraction. Wundt: "Mechanik du Nerven," 1871.

place during a much longer period of intra-uterine life. This shows most distinctly that the nutrition of central nervous tissues is greatly aided by sensory stimuli." "The centripetal nerve-tracts are the keys which start the mechanism of the entire central nervous system; and we see that the peripheral nutritive influence is indicated by the special order in which nerve-tracts acquire their white substance" (Meynert, "Psychiatry," Transl. Sachs., pp. 268 and 269, 1885).

As far as the sensory centres are concerned, there is no indication that their storage capacity in hysteria is deficient; indeed, the preponderance of sensation over centrifugal force, motor or mental, would lead us to infer a relative excess of storage in these centres. But the theory of deficient storage of force in hysterics is based on their inability, as compared with persons soundly organized, to bear fatigue, mental exertion or emotion, or privation of food, or fresh air; peculiarities which are noticeable even in persons who, at the time of trial, are in good health, with their constitutional tendencies latent.

Exertion, mental or physical, implies nervous discharge; the capacity for this is proportioned, partly to the amount of force-material previously stored in nerve-cells, partly to the capacity of these to rapidly store up new material, even while discharging-processes are going on. Deficiency of storage necessarily accelerates the moment when consumption of force-material must be complete, unless this can be rapidly renewed — hence accelerates the approach of fatigue. But the same deficiency in storing processes, which would lower the amount of accumulated supply, might be expected to render storage during action difficult or impossible. The hysteric, therefore, should require more absolute

rest for recuperation after exertion than is necessary for a normal person. The same circumstances would render either privation of food or of abundant air-supply sooner intolerable. Hence it is the hysteric who is most likely to faint in a badly ventilated room.

The portions of the nervous system whose capacities are impugned by these facts, are those which are associated with centrifugal impulses or with the liberation of energies in action. These centrifugal energies are of two kinds : motor and mental. The stimuli which provide for the storage of force-material in the nerve-centres concerned in mental and in motor action are more indirect than the stimuli of the sensory centres. It seems to be the discharge of motor energy, which, possibly by emptying the cell of a certain amount of material, principally determines the acquisition of new material from the blood, and its storage in forms of higher complexity. It is well established that the nutrition of the muscular fibre depends largely upon muscular action, which involves repeated explosive decompositions and elimination of material from storage cells. In centres of incessant reflex action, this stimulus from its own function may be supplied as constantly as is that of the sensory centres. But wherever volition is involved there exists the possibility of avoiding action and, by so much, of lessening the amount of stimulus which should be supplied to the motor mechanisms.

In the cortical motor centres of the brain, according to the bold and ingenious hypothesis of Meynert, a second form of stimulus exists, derived from the registration of impressions during the performance of reflex-motor acts by subcortical mechanisms. Of each such act an impression or image is said to be registered

upon the cortical cells connected with the nerves supplying the contracting muscles. The impression is called by Meynert an "innervation sensation," and is supposed by him to be transmitted to the thalamus from nuclei of the tegmentum, which are themselves connected with the lenticular body, and from the thalamus to the cortex, by the fibres of the corona radiata. The cortical cells are thus rendered "spectators" of all reflex acts. The registration of the impression is attended here, as elsewhere, by chemical synthesis, *i. e.*, by the storage of material destined for the future elaboration of force.

The sensory impression which has initiated the reflex act is also registered in the cortex, and, when transmitted to other cortical areas associated with that which has received this impression, it becomes transformed into a secondary, that is, into an ideal impression. The revival of such a secondary impression, thus the memory of the original sensation, tends to revive the image of the motor act originally associated with it, and which has been registered in the motor centres. The revival of this image liberates energy in a centrifugal direction, along the fibres of the pyramidal tract to spinal nerves and voluntary muscles. This energy, due to intracerebral stimulus, is said to be voluntary; but it is directed to the same nervo-muscular mechanism as had already accomplished motor acts of the same form as those now designed, *i. e.*, the subcortical reflex mechanisms.[1]

Thus the stimuli to storage in the cortical motor

[1] Meynert, loc. cit., gives a diagram, illustrating this theory. It shows the conversion of a reflex movement of withdrawing the hand from a candle by which it had been burned, into a voluntary movement, determined by recollection of the burning sensation acquired on a previous occasion, and itself revived by a new sensation—the sight of the candle.

centres are: 1st, the registration of impressions of innumerable reflex motor acts, performed involuntarily, and before conscious volition is possible; 2d, the liberation of energy under the influence of intracerebral impulses associated with memorized and with secondary impressions. This latter constitutes the performance of voluntary acts, or in other words, is the exercise of the positive function of the centre, the evolution of its positive work.

The "latent areas" of the cortex (Exner), unconnected with either motor or sensory tracts, continually receive, through association-fibres and the gray network of the surface, secondary impressions obtained by revival of those which had been originally registered in the motor and sensory regions. Consciousness is gradually built up of masses of such secondary impressions, and thus is finally traceable to memories of the impressions made on the organism by the outside world, and of the movements which have been performed by the organism in direct or indirect response to these impressions. It is to be presumed that the registration of the secondary impressions is attended by chemical synthesis in the registering cells, similar to those which accompany the registration of primary impressions.

And further, we must infer from the foregoing considerations, that each ganglionic cell or area of the cortex acts both as a receiving and as a discharging centre. In registering sensory, motor, or revived (ideal) impressions, its protoplasm performs a work of chemical synthesis. In transmitting impressions, either in a centrifugal or in an intracerebral direction, the same protoplasm effects a chemical decomposition, whereby energy is liberated.

The same reasons which compel us to infer that, in motor areas, this liberation of energy acts as an indirect stimulus to storage, apply to all the areas of the cortex, to the latent (mental ?), as well as the rest. Thus the intracerebral circulation of impressions is *the* stimulus, although an indirect one, upon which force-storage in the cortex depends, and for those cortical regions which receive no direct stimulus through centripetal nerves, and no indirect stimulus through motor nerves, this intracerebral circulation is the only functional stimulus. When an impression is received on a ganglionic cell, its registration directly effects chemical synthesis ; when an impression is liberated, its transmission indirectly does the same thing.

The smaller the amount of force-material stored up from the blood under the influence of the centripetal stimulus, the smaller can be the amount of work evolved in the centrifugal direction in a given unit of time. If the amount is increased, the period of its evolution is shortened—*i. e.*, the advent of unconquerable fatigue is accelerated. Conversely, when we note the speedy advent of fatigue, as in children, women, and hysterics, we must infer that the storage of force-material has been less than in cases where the period of exertion can be prolonged. The wide diffusion of the "hysterical temperament" in women is correlated with their generally lesser capacity for the storage of force, which may, nevertheless, remain within physiological limits. If the deficiency fall below these limits, it results in the altogether morbid limitations of hysteria. And, while there are many women whose capacity for force-storage — as measured by their capacity for exertion—considerably transcends the average of their sex, and even reaches the masculine average, so there

are not a few men whose capacity in this respect falls to the level of hysteria, and who exhibit hysterical phenomena in consequence.[1]

The sensory centres of the brain in hysterics are exposed to two different kinds of derangement — corresponding to two opposite phenomena — anæsthesia and pain. In the first, the registering power of the centre is so diminished as to fall below the level of consciousness; and this either by privation of blood-supply, through vaso-motor spasm, or by direct depression of protoplasmic energy to appropriate material from that. The latter case enters completely into the general theory of depressed storage-power in hysterical nerve-tissues.

In the innumerable forms of hysterical hyperæsthesia, an opposite process must occur. The centripetal stimulus remaining the same, it would seem as if a larger amount of chemical syntheses were effected in the centre under its influence. When a sensory centre is subjected to an excess of stimulus — through a violent centripetal irritation — it is known that the functions of other nerve-centres may be transiently arrested or inhibited. We may ask whether the same inhibition of other centres is not liable to occur when in the centre there is an excess of reaction to a normal stimulus?

Inhibition has been explained as a phenomenon of interference between the waves of molecular movement transmitted along nerve-fibres; interference analogous to that, which, when occurring between waves

[1] Charcot remarks that male hysteria has become rather a topic of the day, "sujet à l'ordre du jour." He cites a thesis by Klim, containing sixty cases, and a monograph by Batault, containing two hundred and eighteen. See Gaillard's Journal, June, 1886. I have seen several such cases.

of light causes darkness, and between waves of sound causes silence.[1]

In the nerve-tissues, if these waves of molecular movement coincide, the intensity of the movement is increased. But if one wave be retarded out of the normal rhythm, so that its crest fall within the trough of the other, this second wave will be antagonized, and the action of the nerve-centre from which it emanates will, therefore, be apparently diminished. In reality it is not the activity of the centre which is diminished, but the effect of that activity.

This is the explanation of inhibition given by Claude Bernard for the chorda tympani, by Ranvier for the vagus, by Lauder Brunton and by Wundt for the phenomenon of inhibition in general. In accord with this conception, we may suppose that when in a nerve mass, as the sensory centres of the brain, the negative work of intra-molecular synthesis and storage becomes greatly increased, the wave of movement constituting the positive work or centrifugal impulse may become retarded, and this disarrangement of normal rhythm may suffice to make it interfere with and antagonize the molecular waves coming from other cortical areas. These latter, therefore, would be inhibited, as an indirect consequence of the surcharge of sensory centres.

The violent centripetal irritations of sensory centres which are accompanied with pain, always tend to arrest motor and mental action. Their action is not limited to the fore-brain; the arrest of the heart's action is a well-known phenomenon of sensory inhibition. Nor

[1] Brunton: "Pharmacol.," p. 200; Ranvier: "Leçons d'anat. gen.," 1877–78, p. 170. Cl. Bernard: "Rapport sur le progrès de la phys.," 1867, p. 67. Wundt: "Allgemeine Nerven Physiologie."

is it probable that the irritation expends itself exclusively upon the sensory centres of the cortex, but is rather distributed throughout all the receiving stations of the cerebro-spinal axis. Now, when under the influence of a normal stimulus, the cortical centres have acquired the habit of registering impressions abnormally and excessively, as is shown by the patient feeling pain entirely out of proportion to the magnitude of the irritations, we must believe that the work of chemical synthesis excited in the sensory centres, is also excessive. It should, or at least may follow, that the waves of molecular movement transmitting impression in an intracerebral direction become retarded, and thus "interfere" with waves coming from other cortical areas. Then, though to a less degree than with violent pain of peripheric origin, the play of intracerebal associations and impulses, and their ultimate convergence upon motor (volitional) acts, will be interfered with. Hysterical "paralysis of volition" should be the necessary correlative of hysterical hyperæsthesia.

Many facts indicate that the inhibition of one nerve-centre by another is powerful in proportion as the storage of force in the first exceeds that of the second. The feeble control of the vagus centre over the heart in rabbits, the feeble control of the cortex over the subcortical motor (convulsive) centres in young children, negatively illustrates this law.

In accordance with this, the inhibition of the non-sensory cortical areas will be easy, in proportion to an habitual deficiency in the storage power inherent in their tissues. And since the active function of these areas in motility, volition, and thought, has been shown to furnish an important though indirect part of

the stimulus upon which this force-storage depends, every thing which diminishes such activity, diminishes the power of resistance of these parts of the cortex to the inhibitory influence of sensory areas—renders the latter, if we may so express it, more and more tyrannous.[1]

It is generally admitted that activity of thought and of motion tends in some way to blunt sensation. We can most clearly represent this fact to ourselves as implying that, during such centrifugal activity, currents of molecular movement set in from the sensory centres, involving liberation of energy from them and chemical decomposition in them—thus elimination of material that had been previously stored up, perhaps in excess. In the primary reflex acts the current of impression always passes from the sensory toward the motor centre. But, as Meynert observes, in the brain, impressions can certainly traverse fibres in both directions ; and as the impulse to voluntary movement does not come from the areas registering the primary sensation, but from others which have become associated with them, it is perfectly in order to suppose that the intracerebral discharge of the sensory areas is often initiated by the play of molecular movements in the mental motor regions. And this brings the argument round to its starting-point, and suggests that one way

[1] According to Ebner and Munk, the sensory areas of the fore-brain are not limited to those portions of the cortex which receive the fasciculus from the "carrefour sensitive," but extend beyond these until they cover all the motor zones as well. It seems not impossible that at least many areas, even many ganglionic cells, should be regarded as sensito-motor, and this especially if the fundamental function of nerve-elements be accepted as "sensitiveness." But the facts, as at present understood, seem to indicate a frequent, though not universal, commingling of sensory and motor elements rather than an identity of these in function. The schema of sensory inhibition stated in the text must, of course, be provisional.

in which the sensory centres may become hyperexcitable, through excess of storage material, is through the lessened discharge of these centres when the play of centrifugal activities is defective. It is certain that the sensory centres of the cortex are capable of continuing their functions of registration almost indefinitely, even in the exaggerations of pain, while the exercise of either active thought or mobility has quite a limited duration.

The foregoing considerations indicate that the following series of conditions succeed each other in the cortex of the hysterical brain: 1. A diffused deficiency in storage power; deficiency shared more or less with other nerve-tissues, and usually congenital, but sometimes acquired. 2. Nevertheless, effecting of abundant storage in the sensory centres, under the permanent, and thus relatively excessive stimulus of centripetal impressions. 3. Deficient centrifugal activities, mental and motor, or exhaustion of mental and motor areas by exertion performed with inadequate storage material. 4. Deficient discharge of sensory centres, which continue to store material under the stimulus of centripetal impressions, but fail to decompose and eliminate this sufficiently when centrifugal movements diminish in activity. 5. Hyperexcitability of the sensitive centres, which contain an excess of force-material produced during registration of impressions, and not broken up by their transmission. 6. Tendency on the part of these surcharged sensory centres to inhibit the activities of the rest of the fore-brain.

The phenomenon of mental inhibition, resulting in inability for mental exertion, is extremely common in hysteria. It is often described as "causeless mental

depression," as "wilful hysterical indolence," or as "brain exhaustion." I have not found the suggestion anywhere, that the depressive mental phenomena of hysteria depend upon functional inhibition of the thought-areas of the brain. Yet this view seems to me the true one, and alone consonant with all the facts of the case. I have often noticed this condition in uterine disease, where it persists until this is cured. It is not by any means always associated with pains, either in the pelvis—the then focus of irritation—or in the head. After the foregoing analysis it may be inferred that, in these cases, impressions have been generated on a diseased endometrium, or among pelvic nerves, which, though not giving rise to local pain, may, when transmitted to the sensory centres of the cortex, so overexcite them that they inhibit the remaining cortical areas.

The following case offers a curious form of cerebral inhibition :

CASE I.— Unmarried woman, a teacher. Subject for several years to attacks of transient amblyopia in the left eye, coming on many times a day, and lasting from a few seconds to a minute or two. These attacks had been diagnosed by two competent oculists as "epilepsy of the retina." During a year before consultation the patient was also subject to nervous attacks, in which consciousness seemed to be, not abolished, but perverted for a while. The condition is imperfectly described by the patient, who can only say that "every thing seems strange," that people do not seem to be the same ; that she looks very badly to them, and has an inexplicable but profound consciousness of distress. This condition may last fifteen minutes, half an hour, or longer. Examination discovered marked prolapsus of the uterus, so that the cervix came just to the introitus, and apparently rubbed upon the labiæ minoræ. There were no local symptoms of the prolapsus. The uterus, which was structurally apparently healthy, was replaced by a cup-pessary, and the cerebral attacks immedi-

ately and permanently disappeared. The ocular attacks persisted. The entire persistence of consciousness during the attacks exclude, I should think, the diagnosis of general epilepsy.

These attacks seem to me to illustrate, though in a peculiar form, and on a transient and intermittent scale, the cerebral inhibition that is so common and so distressing in hysteria, perhaps especially in that of pelvic origin.

Such inhibition, I would suggest, is the real basis of the mental symptoms, which result, not from excess of mental exertion, but from peripheric irritations in predisposed persons.

The sense of mental inability is usually attended with psychic pain, and the latter is sometimes so predominant that the former is not complained of. Psychic pain, if we accept Meynert's exposition of it, is a direct consequence of cortical inhibition.[1] Whatever interferes with the free diffusion of functional activities throughout the cortex, and with the localized hyperæmias attendant upon these, occasions the "hampered mood," which expresses itself as mental distress, or psychic pain. The immediate mechanism of this is the same, whether the cause be physical — *i. e.*, hysterical — or moral — *i. e.*, objectively justified by events. Meynert thus describes the latter form of inhibition:

"The news of the death of a person who was bound up with a good portion of our thoughts, whose image would be frequently revived in our brain by the most manifold associations, and which, when presented to the brain, would arouse all sort of secondary presentations and pleasurable emotions — such news, we repeat, would cause inhibition of all these associations; and the place of easily excited associations will be usurped by others not yet easily transmitted. Inhibition is attended by emotion and psychical pain."[2]

[1] Loc. cit., p. 193.

[2] Loc. cit., p. 103.

According to the same author a second condition exists, which must constantly tend to increase both psychic pain and mental inability whenever cortical activity is diminished or inhibited.

During the functional activity of the cortex, or of any segment of it — and it is probably never active throughout its whole extent at once — the vaso-motor nerves of the arterioles going to that segment are inhibited, the blood-vessels consequently dilated, and at the same time a direct attractive force is exercised on the blood-current by the chemical processes which are quickened in the ganglionic cells. As a consequence of this combined effect, a larger current of blood is carried to the active tissue. Conversely, when the cortex ceases to be active, as in sleep, or from being itself inhibited, or under any other influence, the vaso-motor tonus of the blood-vessels is resumed, the blood-vessels contract, the cortical tissue becomes relatively deprived of blood—anæmic, or, to use Meynert's expression, dyspnœic. This condition again tends to diminish the power of functional activity in the cortical segment or segments to which the contracted arteries are distributed.

The foregoing considerations may explain the phenomena of mental depression (inability for exertion, psychic pain) both in grief and hysteria. In the former the activity of more or less extensive areas of the cortex is directly arrested by destruction of the objects and associations which call this into play. In the latter the same activity is inhibited by the excessive activity of the sensory areas. In both cases the diminution of functional activity in the ganglionic cells of the cortex is followed by an excess of activity in the subcortical vaso-motor centres released from cortical

control. Hence, in the corresponding segments of the cortex must follow localized anæmias, which tend still further to hamper the functional activity of these segments. The greater and more unimpeded the functional activity of the cortex, the more widely diffused the attendant hyperæmias, the more intense is the consciousness of physical well-being or happiness. The unimpeded diffusion of intracerebral impressions irresistibly suggests a correlatively facile diffusion of desire and activity over all impediments in the outside world; consciousness is permanently triumphant. In the contrary case the arrest of cerebral activities suggests as irresistibly oppression, defeat, humiliation, disaster in external events; imposes subjectively the depressing emotions of mortification, distrust, and apprehension—the depression of spirits which is unconquerable, even when the patients themselves recognize its objective groundlessness. "I have every thing to live for, but I am perfectly wretched," is a common remark. "I know I am better, because I can now look at that undertaker's shop on the corner without feeling ready to burst into tears," remarked one patient to me.

This depression often reaches its maximum during pregnancy, when hysterical women often say they will "go crazy," and not infrequently commit abortion, only to rid themselves of this subjective misery.

When the personality is so completely invaded that the patient does not recognize the groundlessness of her mental suffering, the case becomes complicated by her endless misconceptions of her social relations. A (relatively mild) form of the delirium of persecution is extremely common among hysterics, even those who never exhibit the severer physical phenomena of the

disease. The harmonious maintenance of social relations seems to demand the self-consciousness of an energetic and adequate personality. Cortical inhibition, which weakens this consciousness and fills it with self-distrust, almost necessarily engenders suspicion of others.

In the typical hysterical temperament egotism is a noticeable feature. In hysterics of small minds this may suffice to exclude all interest in external objects. In larger and more cultivated minds such interests are not excluded, but there is an extraordinary tendency to look at them only in their relation to the person, and only in so far as they can be made material to subserve his or her vanity and *amour-propre*. This remarkable tendency is clearly traceable to the predominance of the sensory functions of the fore-brain. Nerve-currents constantly direct attention toward the goal to which they flow. For centripetal sensory impressions this is the receiving organism ; for centrifugal, it is the world upon which that organism expends its energies. Exaggeration of the sensory functions constantly tends, therefore, to exalt the consciousness of the personality over that of the external world. Activity of the voluntary functions constantly tends to divert attention from the personality to the external world. When this habit is firmly established, feelings, as well as actions, direct attention to the external world in which they originate; the individual constantly becomes more and more objective. On the other hand, the person who, in the presence of interesting or impressive events, is only preoccupied with the emotions or sensations they may have engendered in himself, is distinctly marked with the hysterical stigma, even though, which is rare, no other sign of it ever appear.

One curious result of the psychic aspect of hysteria is the manifold way in which it checks the development of the maternal instinct. The frequency of uterine disease in hysterics — the frequency with which their reproductive organs are imperfectly developed, the frequency of accidental abortion — entails sterility in an immense number of cases from physical causes. When hysterical women bear children, they are usually unable to nurse them. In cases where there is no physical impediment to conception, this is often purposely avoided from mere moral perversity. The patients profess to hate children, are in despair if they become pregnant, and, as already noted, not infrequently commit abortion, under the influence of the intense mental depression to which a pregnancy subjects them. When such women nevertheless have children, the hysteria, if not too profound, may be cured. But not infrequently the defect in maternal instinct persists, and the lives of the children are made wretched by the ceaseless exactions, and even increasing selfishness, of the hysterical mother — personal selfishness which is in unnatural contradiction to the profounder maternal egotism which is natural. These conditions are no more universal, or all combined in one person, than are any other symptoms of hysteria. Those women who are sterile from physical incompetence are often tormented all their lives by the longing of unsatisfied maternal instincts. Many hysterical women do make devoted, though rarely judicious mothers. But if not for one cause, then for another, the net result is a great diminution of complete reproductive capacity in hysterics.[1]

[1] The classical notion that sexual impulses are particularly strong in hysterics is certainly erroneous. Both physically and morally, these are often either sin-

The bearing of children implies the liberation, on an immense scale, of centrifugal energies, mental and motor. It is the type of an action—correlating, correcting, and balancing—a feeling, emotion, and passion. From a philosophical point of view, therefore, the sterility or the deficient maternal instinct of hysterics belongs to the same class of conditions as have been already described, and in all of which there is deficiency of motor (centrifugal) force, with conservation of sensory (centripetal) function.

The physical sterility, when congenital and not acquired, allies hysteria, even when remotely, with the neuroses of degeneration. What I have termed the moral sterility, which, in one way or another, results in perversions of the maternal instinct, can be traced to the same preponderance of sensory functions, with exaltation of the narrowest nucleus of the ego, that, namely, which is constitued by the limits of the physical organism. The normal maternal instinct implies one of the first and always the most powerful enlargement of this nucleus, so as to embrace the offspring within the pale of self-consciousness. Failure of this instinct implies a most unnatural narrowing of the range of life within the sensory or purely personal sphere.

Most important, both for diagnosis and for justice, is it to recognize that the mental and moral defects which result from the conditions described are by no means always present. To many hysterics may be applied the phrase reserved by Clifford Albutt for

gularly deficient or singularly perverted, the latter trait constituting one of the first links with insanity. The peculiar whims in these respects of hysterical women often add to their tendencies to sterility by leading them to avoid marriage. Molière has drawn a truthful picture of the refined hysteric in "Les Precieuses."

"neurasthenics," whom he would distinguish from them,—"they are the salt of the earth."[1]

Just as marked intellectual ability, and even genius, is quite possible in hysterics,[2] so may the most amiable, unselfish, and affectionate character be not infrequently found among them. These facts simply mean that the organic tendency, though existing, has been counteracted, either by a development of cortical tissue considerable enough, and endowed with sufficiently abundant associations, to resist complete inhibition in mental spheres; or else by the educational direction given to the formation of associations, and to habits of action, which enables these to offer resistance to sensory inhibition.

Between the cases where mental depression is caused by sensory inhibition and those where it is due to the inhibition of associated ideal impressions, lie the others, where a real moral cause permanently deranges the mental mechanisms, and the affected persons become hysterical from grief or shock. These cases are in many respects analogous to cases of chorea from fright. An impression is made upon certain cortical areas so powerfully that they remain over-excited, and inhibit the activity of the rest. In chorea it is the motor regions of the cortex which are chiefly affected by the inhibition. In adult hysteria it is all of the fore-brain which is concerned in thought or volition; the convergence of intracerebral impressions upon centrifugal tracts is impeded, so that thought and volition are held in abeyance. Sometimes even por-

[1] Visceral Neuroses.

[2] Madame de Stäel indulged in the most violent outbursts of hysterical emotion; Charlotte Brontë suffered from prolonged hysterical hypochondria, probably due to endometritis; George Eliot was the victim of hysterical headaches, and probably of other forms of the disease.

tions of the sensory centres are involved in the inhibition; the patient suffers amblyopia, or localized anæsthesia of some sphere of common sensibility. More often the sensory centres remain intact amidst the depression of all the rest, and the patient becomes the victim of agonizing pains—though in the absence of any peripheric cause for pain. These are the pains of cerebral origin, which are typically hysterical.

In suspensive or cataleptic hysteria, which is more frequently induced by moral than by other causes, the entire fore-brain has lost its susceptibility to stimuli; hence has lost its power of either storing force or of liberating energy. The complete suspension of function in these cases is only the maximum exaggeration of the condition which is fundamentally characteristic of all forms of hysteria.

In these suspensive forms of hysteria the perversion of oxidation processes is also exaggerated to a maximum. The amount of urine and of urea is greatly diminished; the latter may fall from twenty to two grammes a day.[1] The phosphoric acid is also diminished. Empereur has measured the absorption of oxygen and elimination of carbonic acid in this class of patients, and has found both greatly diminished. In one case the movement of disassimilation, as thus estimated, was twenty-four times less than normal. According to the same author, cataleptics absorbed more oxygen than they eliminate carbonic acid, although both processes are greatly diminished in intensity.

The extent of these chemical alterations indicate that the depression of function extends beyond the fore-brain, and probably involves the entire nervous

[1] Fabre: De l'Hysterie Viscerale.

system; hence affects all the nutritive processes under its control. Since the main object of the absorption of food, of the circulation of albumen and of its oxidation, is the maintenance of energy in the nervo-muscular system, the suspension of such energy is naturally followed by depression to the lowest point of nutritive absorptions and oxidations.

In chloro-anæmia, the peculiar neurosis of puberty, which is so closely allied to hysteria and so frequently passes into it, the characteristic alteration of the blood has been shown to be a diminution not in the number of the blood-corpuscles, but in the hæmoglobin they contain (Gowers). There is, then, in these elements, a deficiency in the power of fixing or storing oxygen, which, demonstrated in them, may serve as an index to a similar (probable) deficiency in the elements of the nerve-tissues. Between chloro-anæmia, the mildest form of the disorder, and suspensive hysteria, the most complete and severe, stretches an uninterrupted series of morbid states.

The existence of psychic symptoms in a case of hysteria, or in the history of the case, is admitted to establish that the fore-brain is then involved in the disease. But in the cases where these are inconspicuous, the participation of the brain is less readily seen, and still less does it appear self-evident that non-psychical symptoms are to be referred to the brain. Thus, though a few writers define hysteria as a disease of the brain,[1] there are more who call it a diffuse cerebro-spinal neurosis, or a neurosis of the vaso-motor system.

The problem should be thus stated: Given a group of sensory, motor, or vaso-motor phenomena, to

[1] Jolly: Ziemssen's Handbook, art. Hysteria.

decide whether these originate in disorders of the medullary or spinal nerve-centres, or whether they are due directly or indirectly to disorders of the cerebral cortex.

Now, it can be shown, I think, first, that in a large group of cases the phenomena in question either are attended by some mental symptoms, or that these have occurred in the history of the patient previous to the manifestations of the physical symptoms; second, that the character of the "physical" symptoms themselves are explicable when referred to the brain, but not when referred, finally, to lower centres.

It is the neuroses which present these two fundamental characters which may properly be called hysterical; and are so even when they have themselves been caused by organic disease in a thoracic or abdominal viscus, or are associated with organic disease of the nerve-centres themselves.

Neuroses which really originate in medullary-spinal centres, though often presenting symptoms which resemble those of hysteria, and sometimes occurring independent of hysteria, in persons of hysterical constitution, require to be carefully distinguished from the hysterical neurosis itself.

The principal non-psychical phenomena of hysteria are, in the motor sphere, paralysis and convulsion; in the sensory sphere, anæsthesia and pain; in the visceral sphere, numerous derangements, traceable to vaso-motor spasm or the spasmodic contraction of unstriped muscular fibre.

That hysterical paralysis is an affection of the cortical motor centres is generally conceded, chiefly on account of the marked influence often seen to be exercised over it by mental impressions. But this is

also indicated by the (frequently) monoplegic character of the paralysis, and by the preservation of nutrition and faradic contractility in the affected muscles. The second character, identical with that of organic cerebral paralysis, tends to exclude the ganglionic centres of the spinal cord, and to establish the probability of the cerebral origin of the disease. The monoplegic form of paralysis is as characteristic for the cortex in functional derangement as in organic lesion.

Such functional cortical paralysis represents the maximum degree of inhibition of the cortical motor areas—of which some degree exists in the majority of all cases of hysteria. When the paralysis involves the nerves of the lower extremity, and utero-ovarian disease coincides, the paralysis is often called reflex, and supposed to be in some way connected with reflex spinal arcs.[1]

But, first, there is no physiological experiment which exhibits paralysis resulting from irritation[2] of the sensory part of a reflex arc, but only excess of muscular contraction—spasm.

Second, cases of paralysis without pelvic symptoms, or ascertainable lesion, entirely resemble those in which these coexist.

Third, paralyses of distant nerves—as of the laryngeal, or paresis of the nerves of the throat—are very common substitutes for paraplegic paralysis, and certainly lie beyond the pelvic reflex arcs.

[1] Brown-Séquard assumed a vaso-motor spasm in the motor ganglia of the cord, dependent on sensory irritation. Leyden has attacked the vaso-motor reflex theory, substituting that of an ascending neuritis, on the testimony of two cases, with autopsies.

[2] Brown-Séquard's experiments consisted in hemi-sections of the cord, which were followed by hyperæsthesia of the same side due to vaso-motor paralysis.

The following cases are illustrations :

CASE II.[1]—Intensely chloro-anæmic girl of twenty-two. Ovarian hyperæsthesia for a year, without tangible lesion of uterus or ovaries. Then suddenly, incomplete paraplegia lasting twenty-four hours. Recovery ; relapse a few weeks later. Paraplegia remained incomplete for several months. Patient began to suffer from severe dysmenorrhœa ; pelvic pains gradually encroaching on intermenstrual period, until life was rendered perfectly wretched by them. Ovary found prolapsed. Paraplegia became so complete that patient could not move toes, and remained so for seven years. Then oöphorectomy was performed by Dr. Mundé, for relief of dysmenorrhœa ; and with no hope of affecting paralysis. In ten days after the operation, patient could move the toes ; in a month, had quite recovered power of walking. Ovaries, to naked eye, said to have been healthy.

CASE III.—Girl engaged in factory work. Incomplete paraplegia, with fixed right ovarian hyperæsthesia ; no dysmenorrhœa ; uterus retroverted ; otherwise healthy. Permanent replacement of uterus had no effect ; galvanism at times entirely restored power of walking ; this again lost.

CASE IV.[2]—This case was diagnosed as true locomotor ataxia in several hospitals, but the ataxic symptoms entirely disappeared after an operation for laceration of the cervix.

CASE V.—Married woman, aged forty. Subject for many years to altercations with husband ; loss of power of walking —*i. e.*, experienced so much pain in walking that she considered herself unable to walk, and took to bed for two years. No uterine disease at all. Recovery rapid after positive diagnosis of hysterical nature of "paralysis."

CASE VI.—Woman, aged thirty-five. Subject for five years to uterine hemorrhages, associated for a year or two with intermittent aphonia. Uterine fibroid sessile in fundus. Removal ; arrest of hemorrhages, but attacks of aphonia continued to recur for a long time.

These cases, varying superficially, resemble each other in the preservation of nutrition and faradic

[1] The termination of this case was observed and reported by Dr Mundé in the New England Medical Monthly.

[2] Reported by me in the Archives of Medicine as hysterical locomotor ataxia.

contractility; indeed, in the absence of all objective symptoms, and the summing up of the disease in the single condition—inability of the will to determine the contraction of certain muscles. To what could this be due but to depression, or inhibition of the functions of the cortical motor centres in liberating energy in motor tracts in response to intracerebral stimulus? In Case IV. alone did the inhibition of the cortical centres seem to be associated with peripheric irritation, for in Case II. the ovaries were reported as normal, and the operation seems to have been successful through removing the stimuli of the menstrual processes from hyperexcitable sensory centres. The sensations of fatigue, of which hysterics complain so much and so bitterly, often represent a minor degree of inhibition of cortical motor centres. It makes no difference how perfectly may be accomplished nervo-muscular functions through the body, if the only conscious spectator of these—the fore-brain—registers them awry.

When the patient is anæmic or cachectic, there is certainly often reason to suppose that the reparative nutrition of the entire nervo-muscular system is impaired. But this is not the case in really hysterical fatigue, which, though just as real to the consciousness of the patient, may coincide with every sign of excellent general nutrition. The intimate process of the phenomenon of fatigue is to-day supposed to be the accumulation within nerve- or muscle-tissues of the waste chemical products of previous exertion. The elimination of these acid excretæ is often interfered with in lithæmia, from the diminished alkalinity of the blood bathing the cells, and into which the acid substances should osmose largely in proportion to

that alkalinity,[1] Hence the frequent muscular pains, aching, and weariness; and, when the condition extends to the fore-brain, the frequent clinical combination of lithæmia and hysteria. High tension in veins and in capillaries must also interfere with exosmosis from cells; hence the low arterial tension of anæmia, which constantly tends to increase venous tension, interferes with the elimination of waste, and tends to prolong fatigue, as frequently happens in anæmic hysteria. But in the brain exist special mechanisms for the removal of waste, which are correlated with the special necessity for prompt and complete removal. And that it is this mechanism which is principally deranged in the fatigue of hysteria is shown, I think, by the peculiarities of sleep in hysterical persons, and their habitual increase of fatigue immediately after the period which should, normally, restore them. The fatigue-products of the brain, if not of all nervo-muscular tissues, are principally eliminated during sleep. This is the reason that the morning urine contains, as Mandel has demonstrated, larger amounts of phosphoric acid than that formed during the day. During sleep, both the breaking down of waste products into their elements and the elimination of these from nerve-tissue occur more extensively. Among nerve-tissues it should be principally those of the fore-brain which is thus refreshed by sleep, since its activity is much the most completely suspended. Now, it is quite characteristic of persons in whom hysteria exists, or is imminent, that they wake in the morning with a sense of physical fatigue, or of mental depression or irritability. Schopenhauer thinks it is one among many proofs of the theory of pessimism, that the happiest

[1] See Ranke: Lebens bedingungen der Nerven.

moment of the happiest life is that of falling asleep, and the unhappiest moment of unhappiness is that of first awakening. This is true when legitimate causes for mental depression exist, and is also true when their influence is simulated in hysteria.

In the nervous system, and especially in the brain, the waste products do not pass directly into capillaries, but into the lymphatic sheaths surrounding the arteries. The circulation of the lymph-current, and its passage from the perivascular to the subarachnoid spaces, is regulated by the pulsations of the brain, or its variations in volume, by which the lymph-spaces are rhythmically compressed. The brain-pulsation is composed of three factors: the arterial pulse-wave, the respiratory wave, and the vascular wave. The respiratory wave results from the aspiration of venous blood from the brain during inspiration, and the obstruction to its flow in expiration. The vascular wave advances like a peristaltic movement, and consists in rhythmic dilatations and contractions of the arterioles, apart from the cardiac pulse, and dependent on intermittent vasomotor influences. This vascular wave is said by Burckhardt[1] to be much more regular during sleep than in the waking period, and constitutes, according to this observer, the principal motor mechanism for removing waste products through the lymphatic products. It is said to give two to six tracings a minute. Its lowest point (I am now quoting from Meynert's citations) corresponds to the contraction, its elevation to the relaxation of the arteries. When the wave begins as systole in the arteries at the base of the brain, this is constricted, and the brain mass at the same time pushed upward with the advancing column of

[1] Ueber Gehirnbewegungen, Mitth. d. Naturf. Gesellsch. in Bern, 1881.

blood; simultaneously, the arteries of the convexity dilate in diastole and receive the blood; the cerebral hemispheres swell, and, being compressed against the rigid skull, compress the roof of the ventricles and compel one portion of the ventricular fluid to escape by the foramen of Magendie, another portion to flow into the veins of the choroid plexus. In the second stage the arteries of the convexity are in systole, those of the base in diastole, causing swelling of the base, which opposes the return into the ventricles of the fluid which has escaped into the subarachnoid spaces, so that this fluid passes over the convexity of the brain, between it and the skull, and enters the great venous sinuses.

If it be true, as is now asserted, that this vascular wave is of more importance in the lymph-circulation in the brain than either the pulse- or respiratory-wave, it is clear that any disorder of the vaso-motor centres which govern it may greatly disturb the removal of waste products by interfering with the normal development of such a wave. If, for example, the normal intermittence of vaso-motor impulses becomes exchanged for a permanent tonus, the diastolic portion of the wave would disappear, and with it the swelling of the hemispheres by which the ventricles are compressed. There would remain the variations in volume due to the cardiac systole and diastole; but in sleep these are reduced to a minimum. Hence in any persons subjected to abnormal vaso-motor irritations must exist an imperfect removal of waste products from the brain during sleep, and therefore imperfect refreshment by the great restorer.

Apart from the foregoing conditions, we may inquire whether the diminution of oxygen absorbed during

sleep—diminution which amounts to twenty-four per cent. of that of the waking hours[1] — is not liable in hysterics to interfere with the oxidation of waste products, and hence with their reduction to the most soluble form. The normal diminution corresponds, of course, to the diminished demand for oxygen force-compounds, which are evidently formed in smaller quantities at night. But the other destiny of oxygen in the nervous tissues is the complete reduction of chemical substances, whose first decomposition was attended by the liberation of energy. Where the habitual supply of oxygen is very near the margin, the diminution during sleep may easily reduce it below the amount at which prompt and effective oxidations are possible. Hence, by a double mechanism, the sleep of the neurotic is liable to be uncertain and unrefreshing; to be tormented by bad dreams, among which are most characteristic those of falling from a height. The restlessness and bad sleep closely imitate that of fevers, where the nerve-tissues are surcharged with their own poisonous excreta.

The curious researches of Anjel[2] on the peripheric blood-flow during brain-activity offer experimental indication of vaso-motor irritation in the brain. In normal persons, during mental activity, the turgescence of the tissues of a limb enclosed in a plethysmograph is found to diminish—presumably from the afflux of blood to the brain.[3] But in neurasthenics, under the same circumstances, the plethysmograph registered no

[1] Voit, Hermann's Handbuch, Bd. 6, i., p. 205.

[2] Archiv für Psychiat., 1884.

[3] Amidon's experiments on localized rise of temperature in the brain during voluntary motor contractions point also to localized cerebral hyperæmias. See Alumni Prize Essay, College Physicians and Surgeons, 1880, Arch. Med. for April.

change. The author infers that from permanent and abnormal excess of tonus in the blood-vessels of the brain the alterations in its blood-supply are less marked, and especially that less abundance of blood is thrown into the brain during its functional activity. Hence more ready exhaustion by this.

The motor derangement of hysteria which is opposed to paralysis and fatigue is convulsion. The co-ordinate character of hysterical convulsion distinctly marks it as cerebral, as effected in the highest reflex—*i. e.*, the co-ordinating — centres, formed by the subcortical basal ganglia of the brain. Thus, while sharing the cerebral origin of other hysterical phenomena, it does not imply a condition of exalted activity of the cortical motor centres, which would contradict the general theory of their condition we have been trying to establish. On the contrary, the excessive excitability and activity of the subcortical motor centres imply diminished control over these by the cortical centres, which normally inhibit them in part. It is well known that hysterical convulsions are often brought on by painful, or even by simply disagreeable, moral impressions.

The following case is all the more worthy of citation, because illustrating hysteria in the male subject:

CASE VII.—Man, aged sixty. Long subject to attacks of co-ordinated convulsions, diagnosed as hysterical by several American and European physicians. On one occasion, after a trifling altercation at table with an old lady, patient withdrew to his room in great offence, and two hours later was seized with severe attack of typical convulsion; the body curved in opisthotomus, then bounding from the bed in clonic spasms, these alternating with fits of sobbing and tears. Consciousness was evidently preserved throughout.

Painful emotion, it has been said, implies inhibition

of cortical activities. The inhibited cortical areas lose their own power of inhibition over the subcortical sensory-motor ganglia. If the total cortical area thus inhibited be large, the negative excitation of these ganglia may be so great that involuntary but co-ordinate muscular contractions ensue (hysterical convulsion).

The tendency to cortical inhibition should be resisted in proportion to the mass of secondary impressions which have been previously organized—in virtue of the chemical synthesis attending their registration—in cortical areas. This theoretical statement agrees perfectly with the observation of common experience, that the liability to hysterical convulsion varies in inverse proportion to the mass of ideas previously organized in the consciousness of the individual. If this be small, a slight degree of annoyance suffices for the convulsion ; but in the contrary case, the phenomenon, when of mental causation, only appears after prolonged and profound disturbance. It is curious to notice, however, that hysterical convulsion much more often appears after slight than after severe moral causes ; the latter seem to arouse impressions that re-enforce resistance to inhibition.

Finally, the convulsion may be spontaneous. Yet, of all hysterical accidents, I think this is most frequently traceable to the immediate influence of moral events ; also, is the most often limited to persons of narrow intelligence. The post-epileptic hysterical phenomena noted by Gowers[1] are not infrequently convulsive. They are considered by this author to mark the advent of a slight degree of brain-degeneration ; *i. e.*, such impairment of cortical power as diminishes cortical inhibition over subcortical ganglionic centres.

A third form of disorder in the motor sphere is, like

[1] Epilepsy.

paralysis and convulsion, common both to hysteria and to organic brain disease—this is contraction or rigidity of muscles. In organic disease this follows upon paralysis caused by lesion of the pyramidal tract. In hysteria the contraction is not necessarily preceded by paralysis, and this circumstance is often the only means of establishing the diagnosis. In organic disease, muscular rigidity is known to be associated with sclerosis of the lateral columns of the cord, or, more specifically, with descending degenerations of the pyramidal tracts. Correlatively with this discovery, hysterical contraction has also been assigned to these tracts. Charcot has even discovered lateral sclerosis in an old woman who was said to have suffered for many years before death from hysterical contraction.

The primary condition in descending sclerosis is the atrophy of the medullary sheaths of nerve-fibres which have been separated from their trophic centres in the brain. Trophic centres are evidently those from which start nerve-currents. The reason why the fibres of the pyramidal tract degenerate after a hemorrhage into the internal capsule is, admittedly, because the passage of nerve-currents through them is interrupted. The same degeneration is observed after lesions of the central convolutions, when, though all the mechanisms of movement remain intact, the mechanisms for conveying voluntary impulses have been destroyed.

Is it not possible that, if these mechanisms be, not structurally, but functionally impaired, as they are in hysteria, and the passage of nerve-currents from voluntary impulses suspended, the nutrition of the centrifugal tracts may suffer in some manner analogous to that by which the medullary sheaths waste in organic hemiplegia, but much less intense? Thence, as a con-

sequence, the rigidity of the muscles connected with these tracts.

Such a sequence cannot be considered inevitable, for there are many cases of hysterical paralysis without contraction, and many cases of contraction where the inability to move the limb begins at the same time with its rigidity. But it is difficult to see how the line of causation can be in any other direction than that indicated.

Anæsthesia, the first great division of sensory hysterical phenomena, can be interpreted in one of two ways. It implies such defective blood-supply to the cortical receiving centres that they are unable to obtain material for the chemical syntheses of registration, though constantly receiving the stimulus of centripetal impressions. The anæsthesia would then be attributable to vaso-motor spasm.

But it is probable that the nerve-elements of the sensory centres may also suffer direct depression of their power to respond to stimulus — depression analogous to that suffered by the motor centres in paralysis. In both cases the depression simply exaggerates the habitual defect in the power of force-storage. Anæsthesia, like catalepsy, belongs to the graver forms of hysteria. The stimulus to sensory registration is so great and so permanent, that in sensory centres the defect is habitually overcome, even when obvious in others. When these also fail it is evident that the defect is unusually great.

Amblyopia is the most serious form of anæsthesia. The following case illustrates the serious difficulty in diagnosis which this symptom may occasion :

CASE VIII.—Unmarried woman. Sufferer from various forms of neurotic disorder for several years. After a period of several

months of unusually good health, seized suddenly with the most violent pain in eyes, occipital headache, vomiting, and amblyopia, which in a day or two increased considerably, but never to total blindness. The pupils were widely dilated and insensible to light. For two days there was rigidity of the neck and some retraction of the head. Pulse and temperature remained normal; consciousness was unaffected. No ophthalmoscopic examination at the time; a diagnosis was made of a basilar meningitis localized around the optic chiasma. The patient, however, began to recover in a week, but remained subject to violent headaches, as indeed before the attack. Some years later this patient had an attack of incoercible anorexia and vomiting, which terminated fatally in ten weeks. At the autopsy the brain, medulla, and cord were carefully and microscopically examined, and not the slightest trace of organic lesion found. The vomiting, though fatal, had evidently been hysterical, the disturbance in the nerve-centres functional.[1]

This termination made it strongly probable that the cerebral accidents of the preceding years, including the amblyopia, had been also functional, hysterical, developed under the same influence — a neuritis of the median nerve,—as seemed to be chargeable with the final and fatal irritations.

In minor forms of hysteria, disturbance of the visual sphere not leading to amblyopia is extremely common. Much of this is due to spasm of accommodation, with spasm of the internal recti muscles, or else to paresis of the same muscles. These disorders will be presently considered.

Of all hysterical disorders, pain is the most frequent, the most distressing, and often the most perplexing, either for diagnosis or treatment. The important characters of hysterical pains are the following: They predominate on the left side of the body; they are

[1] An organic cause for this disturbance existed in the periphery of the nervous system, in a neuritis of the median nerve. The terminal history of this case has just been reported by Dr. R. Osgood Mason, Am. Journ. Med. Sciences, July, 1886.

entirely out of proportion to the peripheric irritation in which they seem to originate, both in intensity and duration; they are capable of surviving the complete subsidence of peripheric irritation; they may exist in the absence of all ascertainable peripheric irritation; they often develop and cease, like other hysterical symptoms, under the influence of moral impressions; they are constantly liable to diffuse from the locality in which they first appeared into others, not adjacent, but often connected with the first by ramifications of the same nerve-plexus. The diffusion, however, easily exceeds these limits, and often is general. At other times, however, pain may remain with the utmost tenacity, limited to a single spot or nerve-trunk for years.[1]

Spots of hyperæsthesia are usually aggravated by pressure; deeper-seated pain is sometimes relieved by it; thus especially in the head, and when seated in the muscles of the back.

The reactions of hysterical pain to electricity are also variable, although, as a rule, galvanism has a surprising effect in dissipating these pains,—at least for a time.

CASE IX.—Complains of fixed pain in track of last dorsal and ilio-hypogastric nerve, and in iliac branch of the latter. This locality is a frequent seat of hysterical pain, with or without distinct ovarian hyperæsthesia, with which the ilio-hypogastric pain is frequently associated. The application of a galvanic current of fifteen milli-ampères, descending from the spinal cord along the nerves, invariably relieved the pain in ten minutes. After half a dozen applications the patient professed herself entirely cured, for the time at least, though the pain had previously persisted with more or less intensity for a year.

[1] Charcot has recently pointed out the error of considering hysterical phenomena to be necessarily fugacious and mobile.

CASE X.—Married woman, thirty-five years of age. Marked hysterical temperament, in the form of emotional excitability. Symptoms developed after a winter passed in nursing a relative, and suffering with much physical fatigue, and also anxiety. There was uterine catarrh of moderate severity; hyperæsthesia, without hyperæmia of fundal endometrium; left ovarian hyperæsthesia marked; ovary not perceptible. No dysmenorrhœa, but subject to violent "bursting" headaches just before menstruation, immediately relieved by flow. During premenstrual week, invariably severe mental depression.

In addition to the headaches and the fixed pain in the ovarian region, the patient suffered from pain in the cutaneous branch of the second lumbar nerve where it passes over the left hip, in the middle gluteal nerve on the same side, and in the left pudic nerve. All these pains, as well as ordinary headache, could invariably be dissipated for several hours, or even days, by galvanism applied with the polar method. There seemed to be no difference between the effect of the two poles. The method was not tried on the premenstrual headache

This patient was subjected to a certain amount of intra-uterine treatment, which was always very perturbating. The patient certainly derived no immediate benefit from this, though immediately after its cessation, and on going into the country, she became quite well. The galvanism, however, retained a permanently beneficial influence, whose duration constantly increased. My present impression is that this treatment would have sufficed, with time, to cure her; but while under my treatment she at one time consulted a prominent gynecologist, who diagnosed endometritis and ovaritis, and advised a six weeks' residence in his hospital. This advice was not followed, but it was about six weeks later that all symptoms disappeared.

CASE XI.—In this case, a girl of naturally hysterical temperament, developed the most marked hysterical symptoms in connec-

tion with a retroversio uteri, some of which persisted, though much relieved, after the position of the uterus had been rectified. Among these appeared a new symptom—pain in a fixed part of the vagina, apparently in a branch of the pudic nerve. This pain caused, for a long time, endless trouble about the pessary, which, however, had evidently nothing to do with it; and was so much aggravated by walking that the patient scarcely took any exercise. The pain was aggravated by galvanism, but yielded to a few applications of iodine, made while the patient was being much benefited from the health-lift.

CASE XII.—A robust young German woman, twenty-eight years of age, consulted for violent pains, which occupied nearly all the branches of the left lumbar plexus, accompanied by ovarian hyperæsthesia, and which had lasted a year. These pains would be subdued during the application of a strong galvanic current, but would return in from five to ten minutes afterward. There was no ascertainable utero-ovarian disease. Hysterical symptoms during the year the patient was under observation, but no history of these could be obtained at first. At the end of a year the patient was in the same condition, and disappeared from observation.

CASE XIII.—Young lady, thirty-two years of age. Lithæmic family history; some relatives with marked hysterical hypochondriasis. Patient herself had had several attacks of hysterical affections of different kinds, and now consulted for a spot of pain in left ovarian region of abdomen, that, at first thought, might have been associated with uterine or ovarian lesion, but which soon showed itself as pure ovarian hyperæsthesia. This pain was relieved by galvanism, but more so and more permanently by faradism, applied externally, and disappeared after a few applications, though it had previously lasted six months.

CASE XIV.—Young woman, about thirty years of age. Marked and peculiar hysterical egotism; complained of a pain in track of right twelfth intercostal nerve, said to have lasted seven years. Said to have been aggravated by exercise taken under advice of physician. This pain was quite unaffected by electricity.

CASE XV.—Unmarried girl, twenty-seven years of age. Pain in left crural nerve of eighteen months' duration, during a year of which patient had not walked at all. Either galvanism or faradism temporarily relieved pain, but did not cure it. Patient subsequently cured by sojourn at Weir Mitchell's hospital, where fara-

dism was applied to every part of the body except the affected nerve.

CASE XVI.—Very delicate girl of nineteen years of age. Two years previously severe chloro-anæmia, with amenorrhœa of six months' duration. Recovery. Then severe moral strain through illness and death of father. Patient profoundly prostrated in strength, though making every exertion; constant fatigue, anorexia, much insomnia, nervous fever, headache constant, with frequent exacerbations. Tonics, given at first by another physician, produced no effect. Headache finally greatly improved by mild galvanic current, nape to forehead, and with labile passes here. Relief persisted for twenty-four hours, and was especially marked to the distress which had existed at the nape of the neck.

CASE XVII.—Markedly hysterical constitution, though of an active and cultivated intelligence and most affectionate disposition. Ovarian hyperæsthesia developed, together with a retroversion of the uterus, immediately upon an arrest to menstruation through a moral shock received while menstruating. Amenorrhœa persisted for a year; then menstruation returned, but was often accompanied by hemorrhage from the rectum. The ovarian hyperæsthesia persisted for three years more, causing almost entire inability to walk. During this period, however, it was always relieved, and seemed gradually to abate and disappear, under the daily application, externally, of faradic electricity.

CASE XVIII.—Unmarried woman, thirty-eight years of age. Many hysterical symptoms. Pain in right knee, developed after slight sprain, and persisting for several weeks. Readily dissipated by a very mild application of galvanism; polar method, anode to knee. Some return of pain cured in same way, as rapid and more permanent cure effected by strychnine.

The following case illustrates the development of pains by moral impressions, in a way that is all the more interesting from the age and sincerity of the patient.

CASE XIX.—Woman, fifty-six years of age. Neurotic symptoms of many kinds for many years. A month after death of husband, to whom she was much attached, and whom she had nursed through a trying illness, patient began to have the most agonizing pains darting all over the body. The pains had lasted a fortnight

when I saw the patient. They rapidly yielded to bromide and valerian, though for some weeks showing a constant tendency to return.

The same line of reasoning which, as I think, establishes the cerebral nature of hysterical paralysis, anæsthesia, and contraction,[1] should assign the far more frequent phenomenon of hysterical pain also to the cerebral sensory centres. "The impressions of the [entire] body are conveyed to the brain by the ramifications of all the nerves and their terminal organs; *mutato mutandis* we may argue that the cerebral cortex is the surface upon which the entire body is projected by means of these nerves."[2] No sensory impression can rise into consciousness until it has been thus projected upon the cortex; conversely, the sensory impressions that exist in consciousness, without any objective justification, can only arise in the cortex. The sensory hallucinations of insanity sufficiently prove that the cortical terminations of sensory nerves, in this case most notably those of special sense, are capable of generating impressions which are referred to the periphery. The pains of pure hysteria can only be hallucinations analogous to those of insanity, and generated in the sensory centres of the cortex. For where else could they be generated?

As in insanity slight lesions of the auditory apparatus may initiate hallucinations of hearing which suffice for a basis to a delirium of persecution; so in hysteria slight, and even physiological, impressions may suffice to initiate hallucinations of pain in morbid sensory centres. The brain-cortex is the only part of

[1] The cerebral nature of hysterical convulsion is not, I think, called in question.

[2] Meynert, loc. cit., p. 39.

the nervous system which possesses the power of immeasurably magnifying an impression, in a way that we can perhaps rudely represent by the action of the galvanometer or the boussole. This magnifying power, and still more the capacity for generating a hallucination of pain in the absence of all irritation, is often clinically interpreted as the "imagination" and "exaggeration" of hysterics. These expressions, which, properly understood, really place the pain on the most profound morbid basis, by referring it to disordered action of the brain, are, singularly enough, often taken to justify a contemptuous dismissal of the whole subject. But what can be more serious than a fact of consciousness which has been produced by illicit means?

The remarkable diffusion of hysterical pains is often interpreted as indicating diminution of resistance in the spinal cord, with consequent irradiation in it of centripetal impressions. But irradiation in the cord does not lead to diffusion of sensation, but to wider response in reflex movement. This is shown in Pflüger's experiment, and probably also in strychnine-poisoning.

On the other hand, a moderate degree of diffusion of impression through the receiving-centres of the brain would cause the excitation of areas belonging to centripetal nerves which terminate on the periphery at some distance from the one originally irritated. The course of centripetal nerve-fibres may be compared to a sheaf, expanding at both ends and compressed in the middle. The separation of the central terminations of nerve-fibres at the cortex corresponds to the much wider separation of the same fibres at the periphery.

By a diffusion of the irritation from a single focus

may be excited any or all of the pains so characteristic of hysteria—the clavus, inframammary, third intercostal, præcordial, epigastric, infrascapular pains; those in the track of the external branches of the lumbar and sacral plexus, the pain over the crest of the ilium, and, possibly, the ovarian hyperæsthesia.

The frequency with which pain is referred to the regions of the lumbar and sacral plexus, even in the absence of any utero-ovarian disease, may be explained, at least in part, by the masses of impressions which are being continually generated at the peripheric expansion of the utero-ovarian nerve during the rhythmic processes of menstruation. The frequency of slight disorders of these processes increases the probability of morbidly affecting the cerebral sensory centres through their medium. But, as will presently be shown, vaso-motor spasm probably plays an important *rôle* in the sensory symptoms referred to the pelvis, notably in the ovarian hyperæsthesia.

Pain in the track of the occipital and trigeminal nerves, the basis of some of the most violent headaches observed in neurotics, is often difficult to interpret. Are these true neuralagias, irritations of the roots of nerves by obscure nutritive changes in their nuclei of origin? It is well known that Anstie explains neuralgic pain by atrophy of the posterior nerve-roots—minor degree of the lesion which causes the pains of tabes dorsalis.

The old and oft-quoted remark of Romberg, that "pain is the cry of the nerve for healthy blood," has led almost to a habit of referring these and other neuralgias to anæmia. They are certainly often associated with lithæmia. Apart from general conditions interfering with the abundance or the purity of

the blood-supply, the medullary and upper cervical nerves are especially exposed to localized anæmias during irritations of the medullary vaso-motor centre. Such irritations are most frequent in hysteria.

But all the foregoing causes produce pain directly; a real change takes place in the sensory roots, or the nuclei of origin of the nerves, which is simply registered by their cortical fibres in the sensory regions of the cortex. When due to vaso-motor spasms, these neuralgias may be indirectly due to hysteria. In other cases they may be simply associated with hysteria. Finally, though there be at present no absolute proof of such an occurrence, there seems no reason why sensory irritations should not diffuse into the cortical areas of the trigeminal and occipital as into those of other nerves, and thus pain be referred to their distribution even when both their peripheric expansion and nuclei of origin were intact.

Pain in the head—headache—can never be the direct expression of irritations of the cerebral sensory centres, for such irritations are always referred to the periphery of the nerves connected with these centres. The location of pain in the head after cerebral irritation implies that irritation has been referred to the ramifications of the trigeminal nerve in the dura mater, or to the branches of the occipital nerve distributed over the scalp.

This pain may originate in several ways. In the first place, typical hallucinations of pain may be generated in the cortical centres for the dura mater nerves, and referred to their periphery, as in hysterical pelvic pains.[1] In the second place, hysterical vaso-

[1] See Fox (Diseases, Sympathetic, chap. on Hysteria) for analysis of the action of the otic ganglion upon the sensitive nerves of dura mater.

motor irritations, generated through lack of cortical control over vaso-motor centres, may cause spasmodic anæmia of the nuclei or spinal roots of these nerves, or diffused neuro-paralytic congestions of the dura mater. Finally, true neuralgias of these nerves from general anæmia, or from lithæmia, may develop in hysterical persons, and associate themselves with typically hysterical symptoms.

CASE XX.—Amenorrhœa and severe headache, almost incessant for two or three years. Frequently paroxysms of neuralgiform pain in nape of neck, and extending forward in track of superficial cervical plexus. These paroxysms always relieved and finally cured by aconitia, which had no effect on the headache at all.

Hemicrania has long been regarded as a vaso-motor neurosis; as such it is sometimes hysterical, sometimes direct, especially from the blood-poisoning of lithæmic indigestion. A number of distressing paræsthesias in the head are most common in hysteria and in uterine disease — the head is too big; is empty, hollow; is burning, etc. Vertical and occipital headache is most characteristic of uterine disease, and of uterine hysteria. A constant, diffused, dull headache is also frequent, and would be best explained by diffused congestion of the dura mater through vaso-motor paresis.

The following case illustrates the mode of development of these cerebral paræsthesias, in a way all the more interesting because it is analogous to, and not identical with cases previously quoted.

CASE XXI.—Boy, aged twelve; mother anæmic and hysterical, father healthy. Said to have suffered during five years from headache; become most intolerable during last two years, worse in the morning. Head seems to patient to be very large, hollow,

affected with constant, diffused, dull pain; this frequently exasperated into violent paroxysms. During last year has great disinclination to walk; feels as if he would fall, becomes exhausted, often with increased pain in head; will stand and hold on to a railing. Was seen by several eminent physicians, being under the care of one excellent neurologist for two years with little benefit. Finally the mother consulted a surgeon, who discovered a phimosis and operated. The boy suffered from violent nervous agitation and headache for ten days, then recovered. The inability to walk was entirely relieved; the headaches markedly so, with progressive improvement.

In predisposed persons depressing moral emotions may suffice to induce headache of several years' duration.

CASE XXII. and CASE XXIII. were both extremely anæmic young women. In each, after severe moral strain associated with disappointment in marriage, almost constant headache; most severe at the occiput, frequently exaggerated into the most violent paroxysms. In one case these headaches lasted seven years; in the other, four or five; yielding to no remedy, but finally to time.

The generation of hallucinations of pain in cortical centres, like the hallucinations of visional and auditory centres in insanity, in the entire absence of alteration at the periphery or root of nerves, would imply that the ordinary impressions which passed upward from peripheric nerve-terminations were registered in excess, on account of the hyperexcitability of the registering apparatus. In a photographic apparatus, rays of light of the same intensity produce chemical decompositions which vary in amount (depth) according to the chemical preparation of the receiving plate, *i. e.*, according to its sensitiveness. This may represent one analogy. The hallucinations of insanity furnish, by another analogy, indications of the truth of the proposition maintained earlier in this paper, namely,

that excitability of the sensory centres is increased in proportion as the functional activity of other portions of the brain is depressed or inhibited.

Visual hallucinations are by no means uncommon in hysteria. Dr. Hammond has referred visual hallucinations to disease of the thalamus, and thinks that they are precursors of a special form of epilepsy, called by the author "thalamic." The numerous connections of the thalamus with the optic tract [1] render extremely plausible the suggestion that a morbid process in this ganglionic mass may generate impressions which shall be referred by the optic tract to the retina. Of such impressions, however, the cortical visual centres must, since they rise into consciousness, be the spectator and registrar. Further, the hallucination is composed of elements drawn from memory, *i. e.*, from secondary impressions previously registered in the cortex. It is certain, therefore, that the cortex is involved in the disorder, even if its original starting-point be in the thalamus. It seems more probable that the morbid impression is thus first carried by fibres of the optic tract to the cortical visual centre in the cuneus,[2] thence "referred" by the usual mechanism of illusion [3] to the retina and outside world. Apart from the coexistence of sequence of epileptic convulsions — or else of proof of organic disease of the thalamus — there is, however, no proof that visual hallucinations originate in it rather than in the visual centres themselves.

The two following cases illustrate the effect of a prolonged excess of sensory impressions conveyed through upper nerve-tracts to the brain-centres :

[1] Through the posterior fasciculus, the pulvinar, and the corpus geniculatus externus.

[2] Exner, loc. cit. Seguin, Journal of Nervous Diseases, January, 1886.

[3] Whatever that may be.

CASE XXIV.—Aged thirty-four. From fifteen to twenty-eight engaged in excessive playing on the piano as accompanist to singing-teacher, sometimes ten to twelve hours a day. Six years ago begun to suffer with nervous diarrhœa, and this lasted a year; still liable to attacks of it. Five years ago began to have distress in nape of neck, and after a month, while playing on the piano, arms suddenly "gave out." The patient was "prostrated" in bed for two months, and has never since been able to touch the piano. Even the placing of the fingers on the keys—or on a table in the attitude of playing—causes sensation of nausea, and of distress at nape of neck. The same is caused by touching finger-tips, which are excessively sensitive. There is no pain in track of the nerves; the morbid response to touch is felt immediately near the roots of the cervico-brachial plexus. There are often sensations of numbness in the right arm, and occasional pains. The act of turning the head or lifting the eyes causes nausea, and even "great anguish." When the nape of the neck is supported, the patient feels "perfectly comfortable." The head is the seat of many distressing sensations, though rarely of distinct pain. It sometimes seems enormously big, sometimes perfectly empty (sensations analogous to effect of cannabis indica). Inability for mental exertion marked.

On one occasion patient had been to the Catskills, and had immediately begun to suffer from "frightful dizziness," and was obliged to leave. On another occasion, at the Clinton Water Cure, had attack of incomplete paraplegia. This was cured by the Swedish movement treatment. During the five years that the patient had been more or less subject to these symptoms there had been many intervals of comparative health, but never of complete recovery.

This case approximated to the great class of functional neuroses which usually express themselves in localized convulsions on the attempt to execute certain co-ordinated movements. There were no convulsions, however, and very little pain; apparently no psychic symptoms, except the inability for mental

exertion. The attack of paraplegia was characteristically hysterical.

The etiology of this morbid state in an excess of function demanding complex muscular co-ordination, together with the predominance of symptoms of nausea and vertigo, and the degree of relief afforded when the back of the head was supported, all point to functional exhaustion—exhaustion of power to store force—of the co-ordinating mechanisms of the cerebellum, probably associated with a similar condition of the thalamus through the medium of the recurving fibres passing between thalamus and cerebellum, through the tegmentum and pons. The cortex of the cerebrum was only occasionally, and secondarily, involved; and, correlatively, hysterical symptoms proper were few and transitory; there was but a slight degree of mental inhibition; no noticeable psychic pain, but, however, an attack of paraplegia. The case is quoted here, not as an example of hysteria, but of a morbid state lying on the border-line of hysteria, but distinct from it.

CASE XXV.—This case has already been mentioned while speaking of hysterical amblyopia. It has just been related in full by Dr. Osgood Mason.[1] A large train of neurotic symptoms, which, after fifteen years, finally culminated in incoercible vomiting and death by inanition, were caused by a neuritis of the median and musculo-spiral nerve. The neuritis was due to a splinter run into the palm of the hand at the age of two and a half years, and not removed until the age of twenty-two, when the first symptoms of neuritis developed after a blow on the hand in which the splinter was imbedded. The patient was operated upon several times,[2] by section of the median nerve for the intense pain in the arm, and recovered from this; so, indeed, that between

[1] Am. J. Med. Sciences, July, 1886.

[2] Once by Dr. Sapolini, of Naples; three times by Dr. Weir Mitchell, of Philadelphia, or rather in consultation with him and by his advice.

1878 and 1885, the date of her death, she had no local suffering. But, as the autopsy showed, the neuritis continued to progress in the central segment of the divided nerve ; the patient suffered from several severe attacks of pain in the neck in the course of the cervical plexus, but much more frequently from violent headaches and a series of disturbances which, as quoted from me in Dr. Mason's paper, I have thus classified :

First period.—Mental depression, with religious excitement, followed by severe facial acne. Duration, four months.

Second period.—Utero-ovarian congestion, followed by retroversion, ovarian prolapse ; then recovery after eighteen months.

Third period.—After three months' good health, attack of pseudo-meningitis, with violent headache, vomiting, profound prostration, retraction head, rigidity cervical muscles, intense vertigo, amblyopia, dilatation and insensibility of pupils, absence fever. Duration, eight weeks.

Fourth period.—Headache, with localized tenderness of scalp, some local rise of temperature at same spot, sensations of bursting in head, nausea, stumbling gait, heaviness in limbs. Relieved by cauterization of head ; then by iodide sodium.

Fifth period.—Severe nervous dyspepsia ; relieved by faradization.

Sixth period.—Mental depression approaching to melancholia.

Seventh period.—Increase of dyspepsia, alternating with headache, vertigo, and prostration, and once transitory diabetes ; finally, the attack of violent dyspepsia in which the patient lost her life.

The autopsy was completely negative of result, except as regards the nerves in the brachial plexus. Signs of neuritis were found high up in the plexus, in the musculo-spiral and median nerve ; but nothing in cord, medulla, or brain. The violent, long-continued, and finally fatal disturbance of the central nervous system was therefore purely functional.

The phenomena of the disturbance were fourfold : 1. Mental (frequent attacks of mental depression bordering on melancholia, and which the patient distinguished readily from the "normal" effects of her prolonged sufferings).

2. Vaso-motor (expressed by the transient utero-

ovarian congestion, the purple redness of face attending the pachymeningitic form of headache, possibly also by the severe acne).

3. Sensory (violent headaches, attacks of numbness in limb, attack of amblyopia).

4. Visceral (gastric attacks, which, once begun, increased in frequency, duration, and intensity until the fatal issue).

Dr. Mason quotes me as ascribing all these phenomena to a series of vaso-motor neuroses. I should to-day look for the fundamental conditions in the brain-cortex, so long the recipient of masses of irritating impressions coming from the diseased nerves.

When, after division of the nerves, these impressions could no longer be referred to their peripheric termination, they diffused into the cortical areas of the trigeminal and occipital nerves, causing the violent headaches referred to their termination in the dura and scalp. As a consequence of this excitation of the sensory centres, followed various degrees of cerebral inhibition. When this inhibition was generalized, the patient suffered from psychic pain. When the inhibition especially affected the visual centre, the patient had the attack of amblyopia coinciding with the most intense mental and motor prostration.

When the cortex was inhibited in the rest of its functional activities, its inhibiting control over the subcortical vaso-motor centres was proportionally weakened, a condition which left these centres to an exaggerated activity.

Stimulation of the vaso-motor centre is a known physiological result of sudden irritations of sensory nerves, and may be inferred to have existed during at least the exacerbations of the neuritis. The symp-

tomatology of the case contained nothing positively indicative of exaggerated vaso-motor tonus. There were, however, many symptoms only explicable by vaso-motor paresis, which rarely comes on except as a consequence of previous excess of tonus.

The final illness, comprised of visceral symptoms, was evidently a neurosis of the pneumogastric. Preceded as it was by the attack of diabetes, it indicated an affection of the medulla, which we may most plausibly, however obscurely, associate with the long-standing sensory irritation of the medullary vaso-motor centre.

The theory of Meynert, in regard to vaso-motor excitation from withdrawal of the control normally exercised by the cortex of the brain, explains hysterical vaso-motor neuroses as none other can. By it these neuroses are linked with the same fundamental conditions as underlie the sensory, motor, and psychic phenomena of hysteria.

Upon this theory (which has been already exposed), and in view of the many facts which justify the localization of hysteria in the brain, the vaso-motor neuroses of the disease might probably be called negative, because due to the withdrawal of the control over vaso-motor centres which should normally be exercised by the cortex. Positive vaso-motor neuroses, on the other hand, are those which are caused by excitation of vaso-motor centres through irritation of sensory nerves. These may be present in hysterical people, but are not the typically hysterical phenomena as are the others.

The irritability of the vaso-motor system in hysterics is indicated by an immense number of symptoms, among which "cold chills" and cold hands and feet,

perhaps alternating with flushing and sweating,[1] are very frequent.

There are three other and more debatable phenomena observable in hysterics—the first occasionally, the other two very frequently—that I think may be also referred to the vaso-motor neuroses. The first of these is transient albuminuria.

Studies upon the albuminuria of fever, initiated by Cohnheim and ingeniously pursued by Mendelsohn,[2] have indicated that the initial event in this morbid process is irritation of the vaso-motor nerves of the renal plexus. In fever, the kidney, as measured by Ray's oncometer, was found to shrink in bulk coincidently with the appearance of the albuminuria. The shrinkage implied diminished blood-supply, which could only be attributed to contraction of the arterioles under the influence of vaso-motor irritation. With this fall of the arterial current and diminished supply of arterial blood, there would be a fall of arterial tension, consequently rise of venous tension, venous hyperæmia, venous malnutrition of the epithelium of the glomeruli, hence albuminuria, as a result of its anoxæmia and perverted action.

If such a train of sequences can be determined by the vaso-motor irritations of fever, it can be so also by others, including those of hysteria.[3]

Case XXVI.— Delicate young lady. Subject to sick headaches, associated with gastric catarrh. The latter was being treated successfully by washing of the stomach, when a severe hysterical attack occurred, with much purely symptomatic emo-

[1] To which Beard has given a special sexual significance.

[2] Prize Essay, Alumni College of Physicians and Surgeons, American Journal of the Medical Sciences, vol. xviii.

[3] Or of lithæmia, as suggested by Dr. Kinnicutt, Archives of Medicine, February, 1882.

tional disturbance. Urine examined during this attack exhibited a trace of albumen; the tension of the radial pulse was high. Pilocarpine was given, and in twenty-four hours the albumen had disappeared, not to return. It had never been noticed before.

CASE XXVII.— Extremely fragile and anæmic girl, with some endometritis and numerous neurotic symptoms, that, however, did not prevent her from engaging in steady work at a government clerkship. On several occasions albumen appeared in the urine, without a trace of microscopical alteration of the latter. A physician diagnosed nephritis and prescribed a vegetable diet, upon which patient did not improve. On one occasion, while on this diet, I found considerable albumen, but nevertheless prescribed a meat diet. Two days later all trace of albumen had disappeared.

These discoveries in regard to vaso-motor irritation of the kidney suggest, by analogy, a vaso-motor explanation for the celebrated phenomenon, ovarian hyperæsthesia. It is certainly not at all impossible that the seat of this phenomenon, as of the extraordinary pelvic paræsthesias which may coincide with it, is in the cortex of the brain, at the final terminus of the centripetal fibres carried in the utero-ovarian nerve. But all of the fibres of the ovarian nerve whose termination has, so far, been traced, pass to blood-vessels. These fibres must have vaso-motor functions, hence be centrifugal in direction, although there are, doubtless, others centripetal and endowed with functions of common sensibility. The facility of vaso-motor irritation in the ovary is obvious. May we assume, at least provisionally, that the vaso-motor nerves of the ovary are habitually controlled by those cortical areas in which the centripetal fibres of the utero-ovarian nerve terminate? Loss of inhibiting power in these areas would be felt as vaso-motor excitation at the point of peripheric origin of that nerve; that is, as spasm of the arterioles of the ovary. The result of such spasm is

a diminution in the amount of normal blood sent to the ovary.

Diminution of the arterial blood-supply causes dyspnœa of nerve-elements so exquisitely dependent upon oxygen; this, with or without consequent venous hyperæmia, must become a source of irritation to the ovarian nerve. This ovarian irritation has been rendered classical by Charcot, as a characteristic "stigma" of hysteria. It undoubtedly often exists in the absence of all appreciable lesion of the pelvic organs. But the pain and the diffused paræsthesias—the feelings of swellings, burning, etc.—do not differ in form from those which coincide with enlargement and prolapse of the ovary, with endometritis or displacement of the uterus. Such lesions, indeed, can only be excluded after a scrupulous local examination. As this was omitted in all of Richer's cases,[1] it is quite impossible to tell from the histories how far the symptoms in these cases were purely hysterical, and how far the hysteria was symptomatic of utero-ovarian disease.

The third symptom to be considered in this connection is amenorrhœa. The discovery of the vascular wave in arteries, which has suggested so many fruitful considerations in regard to the arteries of the brain, may be applied to those of the uterus also. It is such a peristaltic wave that should propel blood through the uterus toward the endometrium during the menstrual hemorrhage. Vaso-motor irritation interfering with the regularity of this wave may determine cramps of the uterine muscle—hence dysmenorrhœa in the absence of uterine lesion,—and is probably the *immediate* cause of such pain even when organic lesion exists, and is the ultimate cause of the vaso-motor

[1] Recherches sur l'Hystero-epilepsie, 1884.

irritation. In the highest degree of such irritation spasmodic closure of the arteries would arrest the flow and cause amenorrhœa. With an exaggeration of the peristaltic wave, which may be compared to the exaggerated peristalsis of the intestine which causes certain forms of nervous diarrhœa, there should be menorrhagia. Tait's ovarian menorrhagia is probably of this description, and the multiplication of follicles on the surface of the ovary observed after it has existed for some time would be the consequence, and not the cause, of the hyperæmia. These two opposite conditions—amenorrhœa and menorrhagia—are mentioned here because both are so extremely common in all grades of hysteria, and because both, by the theory of the vascular wave, are traceable to derangements in the vaso-motor apparatus of the utero-ovarian system. Either the entire series of vaso-motor centres, or, more especially, that located in the lumbar cord is at fault.

The over-excitability of such vaso-motor centres, which, on this theory, should exist in many cases of amenorrhœa, would be explained by Meynert's theory of loss of control over them when the activity of the brain-cortex was enfeebled. Thus would be explained the frequency of amenorrhœa in hysteria and in melancholia, where it is, indeed, the rule, and is associated with many other signs of vaso-motor disturbance. Kraft Ebing enumerates tendency to amenorrhœa among the signs of the neurotic constitution which constitutes the predisposition to insanity.[1]

CASE XXVIII.—Girl, nineteen years of age. Subject to periods of amenorrhœa, lasting six months at a time. As soon as the amenorrhœa began patient fell into a state of mild melancholia, which lasted until menstruation returned. During three years

[1] "Psychiatrie," Bd. i.

the menstruation, once arrested, did not return, except under the influence of a sea voyage, which after a while was regularly resorted to whenever the menstruation ceased.

The melancholia was attributed by a distinguished gynecologist to the amenorrhœa. It is much more probable that the amenorrhœa resulted from negative vaso-motor excitation, due to loss of cortical control through hysterical cortical inhibition, the latter being the immediate cause of the psychic symptoms.

CASE XXIX.—Girl, aged twenty-two, who until twenty only menstruated once or twice a year, and had been the subject of several hysterical disorders. The last was an hysterical arthralgia, which confined her to her room for an entire year. During this time, however, the patient was, apart from the arthralgia, perfectly well, free from headaches, and menstruated regularly. A few months after recovery from the arthralgia she received news while menstruating which caused a severe shock and moral distress. The flow was at once arrested, and the patient began to suffer from rather severe pain in the left ovarian region. Two or three weeks later the uterus was found retroverted, the left ovary accessible and tender. The physician consulted replaced the uterus and applied ice over the ovarian region, with a view of lessening ovarian congestion and thus restoring the menstrual flow. The theory of the treatment was erroneous, and the treatment certainly unsuccessful, for the amenorrhœa persisted for a year, accompanied by almost entire inability to walk. Menstruation finally returned after a visit to Franzenbad. The ovarian hyperæsthesia and pain on walking persisted for three years longer, then the patient entirely recovered.

Recovery in such cases, as well as the previous occurrence of regular menstruation, proves that the amenorrhœa cannot be due to hypoplasia, or defective development of the aorta or pelvic arteries. In another place [1] I have endeavored to set forth a special view of menstruation, which claims that the blood lost in the menstrual hemorrhage is determined to the

[1] American Journal of Obstetrics, 1885, 1886.

pelvis by the rhythmic growth of the great utero-ovarian plexuses in which it accumulates. This view is not, however, at all incompatible with such a function of the uterine arteries as has here been suggested in the discussion of their peristaltic wave. For, while the accumulation of blood in the peri-uterine veins is the necessary preliminary, as I have claimed, to its evacuation on the free surface of the uterus, a rise of tension in the uterine arteries has been shown to be necessary to initiate the flow through the rupture of endometrial capillaries (Leopold). A rise of arterial tension has been demonstrated to precede menstruation, first, I believe, by myself, but afterward by a pupil of Hegar's, Reine,[1] and by Fancourt Barnes, of England. In such an increase of tension, in which, nevertheless, normal rhythm was preserved, it is probable that a peristaltic vascular wave would be intensified, and blood aspired more abundantly to the uterine arterioles at the same moment that it had reached its maximum of accumulation in the peri-uterine veins.

I think the hysterical character of at least many cases of amenorrhœa is often overlooked. I have myself often mistaken it, until the prolonged observation of the patient has detected the successive evolution of many undoubted hysterical symptoms. It then becomes clear that the amenorrhœa is itself an hysterical neurosis. It is putting the cart before the horse to think that these symptoms are the *consequence* of the amenorrhœa. As well say that acute melancholia was such a consequence; and, indeed, gynecologists are not infrequently guilty of the latter absurdity. The pathogeny of the cases in question, moreover, is made very clear by the fact that hysterical

[1] See Volkman's Klinische Sammlung.

symptoms may be discovered, if looked for, in the history preceding the amenorrhœa, as well as in that following it.

It is not claimed here that the vaso-motor spasm which is suggested to explain the absence of the menstrual flow, and consequent arrest of other menstrual processes, is necessarily the only disorder at the basis of the amenorrhœa. There may be some direct influence of the nervous system upon the processes of reproductive growth at the endometrium, ovaries, and plexuses. But of such direct influence of the brain upon growth there is at present much less known than of its indirect influence through the induction of vaso-motor spasm; and the latter explains the phenomena. The numerous vaso-motor or sympathetic nerve disorders which accompany hysterical amenorrhœa, and which are absent in purely anæmic or cachectic amenorrhœa, are not consequent upon, but co-incident with, the disordered uterine function. They are the common expression of the same fundamental cause.

Case XXX.—Unmarried woman, aged twenty-six. Began to suffer from dyspepsia, and simultaneously to exhibit profound depression of spirits and hypochondriacal preoccupation about her health. While at Salisbury's Health Establishment, following rigid diet for dyspepsia, menstruation ceased, and remained absent for two years. Persistence of dyspepsia, very severe for eighteen months, then relieved considerably by diet and stomach-washing. Then treatment by health-lift, and soon improvement in dyspepsia. In two months menstruation returned; coincidently dyspepsia disappeared, patient felt quite well, and able to eat ordinary diet. A month later symptoms of approaching menstruation occurred, but there was a delay of a day or two in flow. A single local application of galvanism (made to cavity of cervix) was followed on same day by flow. Previous to use of the health-lift, electrical applications had caused nausea without having the slightest effect upon menstrual symptoms.

CASE XXXI.—Well-developed young woman, aged twenty-five. No appearance of anæmia, but to hæmitametre blood-corpuscles always below four million ; hæmoglobine, sixty per cent. Menstruation always irregular, often at intervals of six months, finally of an entire year. During seven years almost constant suffering from headaches of all description. For a year, ovarian hyperæsthesia, aggravated by walking. Numbness and inability to use arms. Often inability to use eyes in reading, etc., for several weeks at a time, though eyes quite free from organic defect. Inability for mental exertion ; frequent attacks of mental depression. Finally return of menstruation—four times in eight months —under combined influence of change of scene and society and use of health-lift. Coincidently headache, much relieved, began to disappear during long intervals ; all other symptoms entirely disappeared.

Spasmodic tonus or contraction of the unstriped muscular fibre of the blood-vessels, has its counterpart in the spasm of other unstriped muscular fibre, which is so common in hysterics. There is the spasm of the œsophagus (globus), of the intestine (cramps), of the uterus (dysmenorrhœa). There is, apart from spasm, the irregular and precipitate peristaltic contractions of the stomach, which result in vomiting ; of the intestine, which result in the well-known nervous diarrhœa. It is generally accepted that both the tetanic spasm and the irregular clonic contractions of unstriped muscular fibre imply an excess of nerve-discharges through the sympathetic nerve, when this ceases to be sufficiently inhibited by the cerebro-spinal axis. The splanchnic habitually inhibits the intestine ; its paralysis is followed both by exaggerated peristalsis and neuro-paralytic hyperæmia.[1]

The facility with which diarrhœa follows, in certain persons, upon depressing emotions, indicates that the inhibition of cerebral activities suffices to remove this

[1] Nothnagel : Studien über den Darm.

inhibitory influence of the splanchnic.[1] *Per contra*, it may be inferred that the splanchnic nerve habitually serves to convey inhibitory influences from the brain.

The constipation of hysterics, which is often so remarkably obstinate, could not, consistently with the theory advanced in this paper, be ascribed to an excess of cerebral inhibition such as causes the constipation of organic brain disease. It is more probably associated with deficient secretion on the intestinal mucosa, through deranged vaso-motor innervation. In severe hysteric attacks the stools, when procured, are apt to be singularly hard, dry, and black. Tetanic cramp, associated with flatulence, interferes with peristalsis.

Spasm of accommodation of the eye and spasmodic action of the muscles of the larynx are phenomena which seem to be intermediate between the disorders of other voluntary and of involuntary muscular fibre.

Hysteric aphonia certainly ranks among the hysteric paralyses, and is, in every respect, analogous to those of the limbs. If, as assumed by Delavan,[2] the cortical centre for the inferior laryngeal nerve be near the centres for articulation, it cannot be identified with them. Paralysis of the vocal cords only by exception accompanies aphasia[3]; and sounds, whose varying timbre would imply varying modulated contraction of the laryngeal muscles, can still be emitted when power of distinct articulation is lost. Conversely, in hyster-

[1] Nothnagel (loc. cit.) comments on the singularity of the circumstance that the large intestine is normally evacuated only once a day. The fact that the typical time for this evacuation is the early morning may indicate that during sleep, when cortical inhibition is reduced to a minimum, the irritability of the intestinal nerves gradually rises until, upon the stimulus of the first meal, peristaltic contractions attain their maximum of power.

[2] On the Cortical Motor Centre for the Larynx, The New York Medical Record, 1885.

[3] It did in Delavan's cases.

ical aphonia the power of articulation with the lips is preserved.

Variations in timbre of the voice are closely associated with the emotions. They vary, also, with the physical modifications of the reproductive organs, and with special mental emotions associated with these.[1] For all these reasons it seems probable that the cortical centre for the inferior laryngeal nerve is more closely connected than the articulating centre with areas for sensory impressions, and especially with the terminal areas for the centripetal fibres of the utero-ovarian nerve. Irritations transmitted in this nerve, or hyperexcitation of its cortical centre, might, therefore, be expected to somewhat especially affect the cortical centre for the laryngeal nerve, altogether inhibiting it (hysterical aphonia), or partially so (causing irregular innervation of the laryngeal muscles).

Spasm of accommodation of the eyes has attracted a great deal of attention lately as a cause of hysterical symptoms, and especially of headache. The neurosis of the motor oculi nerve, upon which such spasm depends, bears a threefold relation to hysteria.

1. It may be due to organic defect of the eye—uncorrected hypermetropia or myopia, astigmatism, etc.—and constitute the intermediate event between such defect and cerebral symptoms.

2. It may be due to a peripheric irritation, and then either coincide with cerebral syptoms simultaneously caused by this, or be itself the immediate cause of them.

3. It may be directly caused by the cerebral conditions of hysteria, of which it is at once an expression and an aggravation.

[1] Darwin points out that the primary use of the voice in animals is for the attraction of the mate.

The following case illustrates the second of these conditions:

CASE XXXII.—A girl of eighteen fell on the end of her back, and soon after developed coccyodynia. A year later, this still persisting, she began to have intense spasm of the internal recti muscles, associated with visual hallucinations and mental excitability of undefined character. The coccyodynia was cured by galvanism, the spasms of accommodation treated by appropriate glasses; the hallucinations and mental disturbance then disappeared.

The numerous nuclei of the motor oculi nerve lie in the gray matter surrounding the aqueduct of Sylvius, and beneath the corpora quadrigemina. Through the medium of the latter they are connected with optic nerve-fibres; hence indirectly with the visual centre in the cuneus. The proximity of this to the terminal areas of the fibres in the sensitive fasciculus[1] may render it peculiarly susceptible to centripetal irritations. Thus in Case XXXII. the irritation caused by the blow on the spine, and, later, the permanent irritation of Luschka's ganglion, must have been conveyed to this cortical region; thence to the visual centre, causing the visual disturbances; thence probably to the third-nerve nuclei, with the effect of exaggerating nerve discharges to the internal recti muscles.

From the foregoing considerations, hysteria appears as one of the most profound and far reaching of all constitutional diseases, or as one of the most serious accidents which can result from utero-ovarian disease or other form of irritation peripheric to the nerve-centres. It is allied to insanity, as being primarily a disease of the fore-brain; and, further, in many of its most peculiar (occasional) symptoms—as amenor-

[1] At the posterior part of the thalamus and internal capsule, turning back to the occipital lobes.

rhœa; or in perversions of fundamental instincts—as that for food, the sexual and the maternal instinct; in its sensory hallucinations, in the predominance of egotistic consciousness over external perception, in its purposeless excitability, depression of effective force, suspiciousness of others, correlative with a sense of personal inadequacy, etc. This relationship of hysteria to insanity is perfectly well recognized by alienists, but often overlooked by the general practitioners or specialists under whose eyes hysterical symptoms usually develop. It is also recognized that hysteria may accompany organic brain disease (Seguin), or epilepsy[1] (Gowers), or precede insanity; in such cases indicating the beginnings of cortical degeneration (Gowers). The relations of hysteria to the degeneration of stock are again indicated by its frequent family coincidence with tuberculosis.[2]

Profound as are the roots of hysteria, and although it be often as incurable as insanity accompanied by cortical wasting, though it be often as tenacious as life, or even occasionally fatal, it is, nevertheless, usually the lighter form of the neuroses and degenerations to which it is allied. It is, moreover, not infrequently symptomatic; that is, though the temperament may have pre-existed, the serious symptoms only date from some curable peripheric or moral irritation.

Is there any distinction to be made between hysteria and neurasthenia? A distinction is often made, based upon the sex and temper of the patient. If this be a female, and notably selfish, the case is pronounced

[1] Apart from the so-called hystero-epilepsy, or hysteria major. Gowers on Epilepsy, p. 175.

[2] Grasset: Brain, 1884. I have frequently had occasion to note this, or the occurrence of hysteria in phthisical families.

hysteria. If a man, or though a woman, amiable and unselfish, the case is called neurasthenia.

It is difficult to see any reason for distinctions on this line; for, as already pointed out, notwithstanding the logical tendencies of the disease toward a most profound self-absorption, these tendencies are entirely resisted in a great many cases, simply because the mass, educated development, and wealth of organized associations in the fore-brain were such that it resisted sensory inhibition sufficiently to maintain, though at expense of much suffering, high moral and mental character.

I think the diagnosis of hysteria rests upon two circumstances: the presence, in the status or in the history of the patient, of psychic symptoms; or the presence of, at a distance from a focus of irritation, symptoms improperly called reflex, and only explicable by the intervention of a cortical arc, either a sensory motor arc, or one exclusively sensory. Hysterical aphonia or paraplegia illustrate the first of such arcs; all "irradiated" pains illustrate the second.

Apart from these cases are others in which the symptoms remain entirely within the sphere of the medulla and spinal cord, which are lacking the distinctive fore-brain symptoms of hysteria, which are usually attended with circumstances of exhaustion of the general nutrition, or localized exhaustion of certain nerves, and for which the name neurasthenia might more justly be reserved.[1]

The cardinal point in the treatment of hysteria should be the constant reference to the cerebral nature of the disease. This recommendation has indeed long passed into common parlance, though based on the

[1] Beard's neurasthenia certainly included hysteria.

most approximate estimate of the cerebral influence. That mental impressions and mental shocks were capable of dissipating entire trains of hysterical symptoms, has often been pointed out. The inference has too often been drawn that the symptoms were "imaginary" and within the control of the patient, while the fact that the imagination, the consciousness, the very citadel of personal existence, has been invaded by a morbid process, cannot fail to threaten paralysis of volition and self-control.

It is in the prophylaxis of hysteria that the widest use may be made of moral impressions, and the most systematic effort made to organize these into the brain as an effective dike against threatened inhibitions. Early in life the ego must be, by habit, "decentralized," until impressions originating externally to the body become as distinct a part of consciousness as those generated within it. Not less important is the cultivated habit of centrifugal impulses to balance the excess of sensibility. Otherwise, though removed from the tyranny of the physical sphere, the organism remains too predominantly under the influence of secondary sensory impressions associated with the emotions. When hysteria develops, it implies that the mechanisms associated with the inmost individuality have succumbed to the accidents and calamities of life. The prophylaxis demands the construction of a personality so robust, the accumulation of resources so wealthy, that every misfortune may be resisted until the moment of real death.

In this connection the words of so distinguished a neurologist as Eulenberg carry all the more weight, because dwelling on facts whose significance seems to be so little recognized among his own countrymen. "The

predominance of hysteria among women," he says, "depends, ultimately, far more upon the social conditions to which they are subjected, than upon uterine catarrhs and erosions. These conditions combine to arrest energy of will and independence of thought in women; to suppress impartial comparison of their own individuality with external objects; to restrain or suspiciously supervise all impulses to free action; and especially to obstruct and oppose any attempt at emancipation from the limits of a narrow and trivial existence. To these circumstances are due precisely the most severe, extended, and incurable cases of hysteria." [1]

Where the social conditions are favorable, the intellect normally active, yet for a time inhibited under the tyrannous influence of a sensation or an association, change of scene certainly has a most extraordinary influence upon hysterical patients. The hyper-excitability of their cerebral sensory centres renders them morbidly susceptible to the influence of visual as of other centripetal impressions, and to the associations generated by these. Hence the material objects in a locality where painful events have transpired constitute real sources of peripheric irritation, incessantly renewing the first. In one case that I knew, a young French lady received, at six o'clock one afternoon, the news of the result of a criminal trial, which, in deciding adversely to her step-father, broke off a project of marriage for herself. Every day for a year at the same hour, when a certain bell struck, the girl had an attack of fever.

In another case (Case XXXIV.), a woman of considerable intelligence, but who, for several years, had exhibited distinct hysteric symptoms, was thrown into

[1] Nervenkrankheiten.

a condition of profound mental and physical prostration by the death of her mother. This condition was complicated by insomnia, and attended by a degree of psychic pain which the patient subsequently described as "the tortures of hell." At the end of two months the symptoms were unabated, and the attending physician began "to suspect organic disease of the spinal cord." The patient was then sent to another city, and immediately began to improve. Without further treatment than a daily séance of faradization, which cured the insomnia, she rapidly recovered.

When change of occupation and change of interest can be added to change of scene the influence is still greater. The more areas of the cortex that can be awakened to functional activity, the more chance there is for resisting sensory inhibition; the more vaso-motor inhibiting power is restored to the cortex, the more the intracerebral circulation of impressions is quickened, centrifugal currents established, and, if we may hazard again the hypothesis already enunciated, the more the surcharged sensory centres may be discharged.

The foregoing influences all bear upon the mental processes of the brain, finally elaborated (it is probable) in its "latent zones." By affecting these, visceral disorders are frequently relieved, not because they did not exist before, but because the visceral functions of the brain in controlling vasc-motor spasm have been indirectly modified, and the disorders resulting from vaso-motor excitation are therefore controlled.

After the psychic, the second great function of the brain which sustains inhibition in hysteria is the motor. Treatment directed to the stimulation of this function may be expected to be beneficial in the same way, and

for the same reason, as the moral treatment briefly alluded to. Many special modes of treatment are already in use for hysterical symptoms, whose real value probably consists in their common power to modify the fundamental condition of hysteria. Thus faradization, massage, Swedish movements, active gymnastics, horseback, and other non-systematic exercise, the health-lift, all energize the cerebral motor centres.[1]

Early in this paper has been quoted Meynert's ingenious theory of the development of the power for voluntary action, through registration in the brain-cortex of impressions or images of movements which have been effected involuntarily, through subcortical reflex arcs. Now, when a muscle is contracted involuntarily by the application of a faradic current, an impression of the movement may similarly be expected to be registered in the motor centres of the cortex. An accumulation of such impressions should so stimulate these motor centres as perhaps to enable them to escape from the inhibiting influence of the sensory areas.

Faradization was first used in treatment of hysterical paralysis. But there is no hysterical symptom to which theory, confirmed by experience, does not show it to be adapted. In the severe Case XXXIV. insomnia was relieved by it, after having resisted poisonous doses of narcotics.

When the current is applied over the surface of the body without contracting the muscles, the effect on the cortex of registering a muscular contraction cannot

[1] Other effects are, of course, produced through the increased inspiration, circulation, and muscular nutrition, but these are apart from the special problem under consideration.

be obtained. The application is, however, often beneficial, except where there is hyperæsthesia of the surface; for this is usually aggravated by faradization. Can it be inferred that in this case the ingoing electrical current, or rather the nerve current it excites, inhibits the excitation of the sensory centres of the brain?

CASE XXXV.—This fragile patient has already been mentioned as the subject of transient albuminuria. While feeling very weak and wretched, with pains in back, bowels, and uterus, she had a séance of faradization lasting half an hour. One electrode was placed at the nape of the neck, the other passed over the abdomen. The patient at once began to feel better, and during two days following was most remarkably improved, all pains disappearing.

Such cases could easily be multiplied. They are in every one's experience, but the results are very variously interpreted. Weir Mitchell[1] only mentions the local effect upon muscles supposed to become better nourished when forced to contract by electricity. The common idea seems to be that the electrical current in some way takes the place of nerve-force when the latter is deficient; an idea that is certainly erroneous. It is true the faradic current can replace the nerve-current in liberating energy from a muscle-cell, just as, under certain circumstances, mechanical stimulus can do the same thing. The improvement of non-paralytic symptoms noticed in the hysterics who do respond favorably to faradization implies, however, central stimulation. The current directly stimulates the negative, indirectly the positive, work of the nerve-centres to which it is brought. The "strengthening" effects noticed, therefore, partly indicate increased negative

[1] Fat and Blood.

work throughout nerve-centres (in the foregoing case, probably chiefly in the medulla), but there is also partly increased positive work consequent on this; thus, in sensory centres, liberation or discharge of energies in a centrifugal direction.

Massage, Swedish movement-cure, and systematic gymnastics are all directed toward the exaltation of deficient motor force. In passive massage, while the surface friction increases the mass of centripetal impressions, in a manner analogous to surface faradization, the passive contraction of the muscles by the movements of the limbs may be supposed to register impressions in the cortex, as in the performance of reflex acts.

When the patient is incited to resist passive movements by voluntary effort, as in the Swedish movement-cure, a higher degree of stimulus of cortical centres is effected. The intracerebral nature of this higher stimulus approximates the action more to normal action. Finally, in active gymnastics, the stimulus is entirely voluntary, the action entirely normal. The method, which cannot be utilized at the beginning of treatment of hysterical paralysis, is inestimable in the treatment of all other conditions. Theoretically, voluntary muscular effort, the physical correlative of mental volition, should be, with it, the cardinal resource in the treatment of hysteria; for it addresses itself to the fundamental condition of the disease—the depression of motor function below the level of sensory function; and it tends to restore the normal centrifugal direction of intracerebral nerve-currents.

I was led to formulate the above statement thus precisely, from observation of the special mode of muscular exercise afforded by the Butler Health Lift. I

first tried this in some cases of amenorrhœa, not at all with the view I at present entertain, but for the purpose of increasing blood-pressure in the pelvis and thus restoring the menstrual flow. The two cases in which this method received a fair trial have already been mentioned.

In Case XXX. the menstruation had been absent for two years, although up to that time it had been quite regular ; the patient was only moderately anæmic, and retained excellent muscular strength, as shown by her capacity to speedily attain a lift of ninety pounds. The patient was also constantly occupied in family duties, especially in waiting upon a sick father, so that moral and even motor centrifugal currents might seem to exist in sufficient abundance. But she suffered from prolonged dyspepsia (though, as shown by the stomach-washing, very rarely from gastric catarrh), from constipation and flatulence, and the psychic symptoms of depression and hypochondria had been extremely marked. It was while these were at their maximum that the amenorrhœa began ; they had much subsided when I first had charge of the case, and the dyspepsia was a good deal relieved by the stomach-pump treatment. This was interrupted, however, and by my advice the patient hired a health-lift and exercised on it regularly. At the end of a month the dyspepsia was much improved, and the patient felt in every respect better. At the end of two months she menstruated. She then was able to return to a mixed diet. The third month there was a little delay, only a day or two, in the return of the menstrual flow ; a single local application of galvanism brought it on. This had previously been tried with no effect but the production of nausea.

In Case XXXI. the first improvement in the patient's health, after three years of almost constant suffering, was observed during a summer spent at Lake Mohonk, where the patient practised rowing. During these three years menstruation had ocoasionally occurred spontaneously, and had several times been brought on by local applications to the uterus of laminaria tents, and iodine internally. These applications, however, often failed, and when they succeeded in determining a uterine hemorrhage, the patient usually felt worse after them, with more headache and prostration. The month preceding the visit to Lake Mohonk an iodine

application had been followed, not by menstruation, but by a four weeks' leucorrhœa. It was noticeable that during this period, as with any other peripheric irritation which lasted a short time, the patient was relieved from headache; but she claimed to feel "wretchedly," and expressed the greatest horror of the experience. In September the health-lift was begun; in October the patient menstruated, exactly a year from the last date. She subsequently menstruated in February, April, June, and July. Coincidently, the headaches greatly diminished in frequency and intensity, ovarian hyperæsthesia entirely disappeared, and the patient felt distinctly and "immensely better."

That in this last case the improvement was not due to the fact of menstruation was indicated by its absence when the menstruation, was brought on by other means. The spontaneous appearance of the flow during the use of the lift was an indication, not a cause, of improvement. The patient always felt better during two or three weeks preceding a spontaneous menstruation, and always worse after one, whether spontaneous or artificial. During the month which followed the second menstruation after the health-lift, the patient was wretched, with severe headache; but after the last two menstruations, remained well.

I think the details show in this case, as in the other, that with the revival of the motor function of the cortex its inhibiting power over the vaso-motor centres was revived, the vaso-motor fibres in the utero-ovarian nerve were restrained from their excess of action, and an arterial afflux of blood permitted to the endometrium. Probably, also, the nutritive processes of growth on the endometrium, the ovary, and the plexus were coincidently permitted to resume their course.

In Case XXXII. there was no amenorrhœa, but the patient, who had been excessively hysterical previous to the replacement of a retroflexed uterus, continued after this to retain some hysterical symptoms. She remained rather weak, and her power of

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the percussion stroke, part of which may be due to increased force of cardiac contraction, but part certainly to diminished tension, as the line collapses (in the first three figures) almost immediately from the summit. The respiratory line rises, showing the increased depth of respiration.

Figs. 3, 4, and 5 (from Case XXXII., with persistent neuralgia in branch of pudic nerve) show a decided increase of tension from use of lift.

Figs. 6 and 7 show a marked development of the percussion stroke and a rise of the respiratory base-line. This patient had a (corrected) retroflexio uteri, and was recovering from a prolapse of the ovary, but was rather free from hysterical symptoms.

Figs. 8 and 9 are from a girl, aged sixteen, who had never menstruated. and was suffering much from headache. The pulse developed considerably by the lift, tidal wave and tension increasing. The girl's health improved very much during the fortnight she used this, coincidently with walking, but she then, for some unknown reason, ceased attendance.

The increased heart-action noticed in all these traces was the direct result of the muscular effort. The collapse of the line in Figs. 1 and 2, as well as the increased tidal wave in the other figures, both imply diminished vaso-motor tension—in the first case simply lowering the resistance, in the other permitting dilatation of blood-vessels.

It is to be noted that if this is not accomplished, and the arterioles remain contracted while the energy of the cardiac contraction is increased, there will be fatigue, cardiac distress, and palpitations. Undoubtedly such cases will present themselves in practice. The danger can be avoided by the careful graduation

of the weight to be lifted, avoiding such as shall too suddenly increase the force of the cardiac contraction.

I have known horseback riding to restore an interrupted menstruation as efficiently as the health-lift. Between the latter, active gymnastics, and horseback exercise, it is possible that there may be little to choose. Still, as with all other remedies, a case which resists one will often be found to yield to another apparently quite analogous. The health-lift is much cheaper than horseback riding, and it has this advantage over calisthenics, that the amount of force exercised is much more independent of the patient's will. When the movement to lift has begun, it must be finished, and with the same weight; must always be performed with the same degree of force. But it is very possible for dumb-bell and other calisthenic exercise to be carried out so listlessly and feebly that no effect is produced at all. All the foregoing methods of motor treatment are much superior to walking; of which, in a great many cases, the patient is quite incapable. Walking may be found to increase ovarian hyperæsthesia, or headache, or backache, or any other symptom. The effect of the health-lift in increasing the force of the circulation, and hence the amount of oxygen carried to the tissues, and especially the brain, may be secured by another physical apparatus, the Waldenburg machine for compressed air. I have obtained the most prompt and marked relief to hysterical dyspnœa and intercostal pains by this method, where there was not a trace of pulmonary disease. One patient purchased an apparatus, and to her daily use of it, for a year, seemed chiefly attributable the relief, not only of the respiratory symptoms, but of many others from which the patient had suffered for seven or eight years.

This winter I have applied the same treatment to an anæmic girl (Case XXXV.), a subject for many years to slight epileptiform attacks, resembling petit-mal, and also to neurotic symptoms, such as Gowers calls "post-epileptic hysteria"; the principal being severe nervous headaches, mental inability, and sense of universal fatigue. The inhalations of compressed air did not diminish the number of epileptiform attacks, though these were greatly controlled by nitrite of amyl. But the improvement in the hysterical symptoms was very marked, so much so that the patient was ready to believe herself on the high road to complete recovery. The relief was always immediate, and especially after a petit-mal attack.

It is to be presumed that the increased amount of oxygen forced into the lungs under pressure relieves the "dyspnœic" condition of the brain-tissues induced by vaso-motor spasm.

The effect of electricity upon hysteric pain has been already discussed. In this discussion has been pointed out the variableness of this influence. This might be inferred from the complex action of electricity, part of which may fall in the desired direction, another part just in the reverse. Thus the passage of the constant current through a nerve tends to lower its excitability, and ultimately paralyzes it; the anode depresses, the cathode exalts, the excitability of same nerve; the centripetal impression sent to nerve-centres first increases their negative work, chemical synthesis, and storage of force; muscular contractions by faradism or interrupted galvanism divert nerve-energy from sensory centres. It is evident that some of these effects tend to antagonize pain, yet the centripetal excitation of the sensory centres should tend to increase

both pain and its cause in the existing hyper-excitation of these centres. Where the latter is very great, any form of electricity does harm. In proportion to the more localized diffusion of the pain, electricity seems to do good, though with many exceptions. For general action faradism is decidedly preferable to galvanism. Galvanism may sometimes locally overcome vaso-motor irritation.

All modern studies of hysteria tend to relegate drugs to the background in the treatment. The array of antispasmodics—musk, assafœtida, valerian, ammonia, etc.—which figure even in Briquet's treatise, are to-day utilized only for occasional and symptomatic treatment. This really still leaves a large sphere of usefulness for these remedies in the management of these often exquisitely unfortunate patients. The treatment of vaso-motor dysmenorrhœa especially calls for suitable "antispasmodic" remedies, while musk has extraordinary value in the attacks of profound prostration which are common. The value of opium in hysterical vomiting, of digitalis in hysterical irregularity of the heart's action, of ergot in the pelvic congestions which so often initiate or maintain hysterical conditions, need only be mentioned here.

The internal use of strychnine should be classed among agents which act directly on the motor system. It diminishes resistance to the transmission of impressions through the cord.

It is a matter of course that the treatment of anæmia, by meat, iron, cold pack, shower bath, mountain air, is often indicated in the management of hysteria, and when indicated becomes of the utmost importance. A richly meat diet is nearly always indicated, for the reason that the ingestion of albumen in abundance is the

most powerful agent for increasing the absorption of oxygen. Yet the same persons who habitually require meat may from time to time require to completely abstain from it for a few days, during attacks of (liver ?) indigestion associated with copious deposits of sand in the urine. There is an hysteria whose basis is lithæmia—form not infrequent in men as well as women. But there is also an intercurrent gastro-hepatic indigestion that seems to be associated with vaso-motor congestions of the liver, and consequent interference with the functions of the gland in the elaboration of urea. It is probable that transient diabetes would often be discovered if looked for, as in the case of severe neurosis already mentioned.

The association of obesity with hysteria is very frequent. Weir Mitchell observes that it is much more difficult of treatment than the hysteria of thin people. The necessity for meat diet, with restriction of liquids, is here as great as in the cases where the obesity is associated with organic heart and kidney disease.[1]

The nutrition of the nerve-centres is impaired in proportion to the deposit of nutritious material in cellulo-adipose tissue, and the permanent hyperæmia of this.

The two following cases strikingly illustrate the effect upon hysterical symptoms—in one case including amenorrhœa—of appropriate diet, with massage and hydrotherapeutic treatment.

CASE XXXVI.—Married, three or four children ; large and very fat woman, weighing two hundred pounds. Since increase of weight, during a year, profound prostration of strength, with hysterical depression, crying, trembling of limbs on walking, palpitations on exertion. Heart probably overlaid with fat ; no other

[1] See Oertel : Therapie der Kreislauf's Störung, 1884.

disease. Placed on meat diet, gluten bread, liquids restricted; tincture nux vomica, cold pack, with massage and cutaneous faradization three times a week. Great improvement in a month. In six months patient had lost forty pounds and was feeling quite well. Diet continued for a year.

CASE XXXVII.—Married woman, aged thirty; three children. Rather short woman, but weighed two hundred and forty pounds. Psychic depression, palpitations, amenorrhœa for seven months. Packs, massage, meat and gluten bread diet. Immediate and striking improvement. Reduction in weight averaged three pounds a week. Menstruated six weeks after beginning treatment, and thence regularly. Spirits improved at once. Treatment continued five months; patient then quite well. At close of year weighed one hundred and fifty-eight pounds.

The *removal of the ovaries* for intractable hysteria is indicated in two different classes of cases: (1) Where their diseased condition is a source of masses of nervous impressions, improperly called reflex, which irritate the sensory centres of the brain and determine the series of consequences which follow on this irritation; (2) where the ovaries are normal, but the irritability of these same centres, acquired in other ways, has become such that the normal impression generated in the menstrual processes causes intolerable irritation. I have known of two cases where Battey's operation was performed with entire relief to an immense train of morbid symptoms, which in one case included eight years' paraplegia. In neither did the ovaries appear abnormal to the naked eye; in one which I was able to examine by the microscope, the morbid changes were very slight.

The first case has been reported by Dr. Mundé,[1] who performed the operation, and has been already mentioned in this paper.

Theoretically, it is perfectly logical, in cases of

[1] New England Medical Monthly.

hyperexcitability of cerebral sensory centres which have resisted all other means of treatment, to remove the ovaries in order to cut off from these centres the large mass of centripetal impressions which reach them when the rhythm of menstrual processes is going on. It has been abundantly shown that this operation is not often immediately successful; either because menstruation persists, or because the nervous phenomena persist in the absence of menstruation. Both, however, tend to subside with the lapse of time, and I think that it is only after two years that we should, if at all, call the operation a failure. I have known several cases where the morbid symptoms persisted nearly to this time, but disappeared afterward.

The facility of abusing the operation is, however, obvious; but the statistical discussion of its value does not lie within the scope of this paper.

II.

TUMORS OF THE BRAIN.

THE symptoms caused by tumors of the brain are due, first, to irritation or destruction of the portions of the nerve-tissue in which they are embedded, or near to which they lie; second, to pressure exercised upon the entire contents of the cranium—nerve-tissue, blood-vessels, and lymphatics. The first class of symptoms are common to tumor, and to all other circumscribed lesions of the same locality, thus especially patches of chronic softening. The second class are common to all conditions in which the intracranial space is encroached upon; such are extra- as well as intra-cerebral tumors, morbid products within the brain, which differ considerably from neoplasms proper, and, finally, abscesses and aneurisms. Thus, the investigation of the case of any patient exhibiting cerebral symptoms demands that we decide: First, whether these are caused by a new growth of any kind, which is encroaching upon the cranial cavity; second, this being admitted, what is the nature of the growth; third, what is its precise locality.

The prognosis must then be framed according to the fact, the nature, and the seat of the growth; and, finally, the (very limited) indications for treatment must be considered.

SYMPTOMS INDICATING THE EXISTENCE OF AN INTRA-

[1] Reprinted from Wood's Reference Handbook of the Medical Sciences.

CRANIAL GROWTH.—These are of two kinds: those belonging to the perversion or abolition of cerebral function, and those indicating a rise of intracranial pressure. The first are the focal, the second the diffused symptoms (Griesinger). It is this second class of symptoms which are of the most importance in distinguishing between tumor and other cerebral lesions, and they may therefore be considered first.

Diffused Symptoms.—These are headache, vertigo, vomiting, general epileptiform convulsions, apoplectiform attacks, psychic disturbances, and choked disk.

Headache is one of the earliest and most constant symptoms of intracranial tumor. It is also often one of the most severe, and, by its persistence and intensity, may be usually distinguished from cephalalgia of other causation. It may precede all other symptoms of the disease for some time, and it is then that some absolutely pathognomonic characteristic would be most desirable, but is hard to find. In a certain number of cases the seat of the headache corresponds to the seat of the tumor; this is oftenest the case with tumors, intra- or extra-cerebral, which occupy the posterior cranial fossa. Even here, however, the pain is sometimes frontal. Localized percussion will sometimes greatly intensify the pain at a particular point; and this may be found to correspond to the seat of the tumor. The headache is often periodic, and then is easily mistaken for an apyretic malarial attack. This is especially the case when a frontal headache seems to imitate brow-ache. On the other hand, occipital headache may simulate the cervico-occipital neuralgia of gouty persons. The diagnosis may be facilitated in either case by careful exploration along the track of the nerve. Nocturnal headache resembles

the syphilitic cephalalgia; and the question is rendered all the more difficult from the fact that brain tumors are frequently of a syphilitic nature.

Although so prominent a symptom when it exists, headache is by no means always present. It was absent in one hundred and forty-eight out of two hundred and seventy-four cases analyzed by Ball and Krishaber. By combining the tables of Ladame and of Bernhardt (the first summing up all cases published earlier than 1868; the second, those between that date and 1880), we can construct the following table, showing the proportion of cases of headache with cerebral tumors of different localities:

TABLE I.

Seat.	No. of cases.	No. with headache.	Per cent.
Cerebral peduncle	10	4	40
Basal ganglia	41	19	46
Cortex	74	37	50
Medulla	28	16	57
Cerebral lobes	196	129	66
Pons	56	37	67
Cerebellum	196	150	77
Corpora quadrigemina	13	9	69

This table helps to confirm the inferences that might be drawn from the physiology of pain. It is rarely to be attributed to irritation of sensory centres; but rather to the stretching of the dura mater, with its rich supply of sensory nerve-filaments from the trigeminus. As some degree of stretching always takes place, no matter what the seat of the tumor, headache is always imminent, but it may not manifest itself if the tumor grows very slowly, and makes way for itself by gradual compression of the brain-substance, and displacement of its fluids. On the other hand, the pain is most certain to occur, and also to be most violent, when the tumor grows in the cerebellum

under the tense fold of the tentorium; it is least likely to occur when room is left for the expansion of the growth at the interpeduncular space. In the pons and medulla, direct irritation of the trigeminus may add a special liability to pain. The great liability to headache with tumors of the corpora quadrigemina may perhaps be due to their intimate connections with the cerebellum. In the cortex, only half the cases were attended by headache; this probably being due to the tendency of the tumor to grow downward, and thus to relieve the tension of the dura. The periodicity in the pain is undoubtedly associated with fluctuations of the circulation, always liable to be diurnally periodic. An initial headache often disappears when paralysis sets in; probably because, by that time, a zone of softening has usually developed around the tumor. The intensity of the pressure is at first either partially or entirely relieved; headache may set in, if the tumor suddenly assume a more rapid rate of growth; it necessarily subsides at the appearance of the terminal symptoms of drowsiness and comatose apathy; the pain being blunted, like other signs of irritation, in the general depression of the sensibility.

Vertigo is a prominent symptom of any organic brain disease, and although frequently present with tumor, is far from characteristic of it. It is probably always associated with direct or indirect irritation of those portions of the brain which are concerned in equilibration; and it agrees well with this presumption that vertigo so much more frequently occurs with tumors of the cerebellum than with those of other parts of the brain. Other "space-encroaching" lesions, *e. g.*, abscesses, so situated as to affect the middle or internal ear, may cause vertigo by the same

mechanism as that which is brought into play in ear disease proper—namely, by excitation of the auditory nerve. As the central fibres of this nerve have been traced to the cerebellum, it seems probable that the sensation of vertigo, whether cerebral or aural in origin, is always finally produced by the same mechanism. The third diffused symptom, *vomiting*, follows the same law of predominance as headache and vertigo—namely, it is much more frequent and severe in tumors of the cerebellum than in those involving some other portion of the brain, with the exception of the corpora quadrigemina, where the liability is at the maximum.

From the following table it appears that vomiting is a much less frequent symptom than headache, but follows exactly the same order of predominance, except in respect to the centrum ovale. This is because the vomiting is partly due to the same cause as the headache—namely, the extreme tension of the tentorium. Extreme tension does not, however, always cause vomiting.

CASE, by King (*Brain*, October, 1882): Two tumors, one on right side of pons extending to left middle peduncle of the cerebellum, the second embedded in the left side of the floor of the fourth ventricle, convolutions flattened, much serum in ventricles, showing extreme intracranial pressure; but optic neuritis developed only after attacks of coma. Headache, but no vomiting.

The immediate cause of vomiting is supposed to be always the excitation of a vomiting centre in the medulla; and this can be brought about by pressure transmitted from any part of the brain. This pressure is, however, more direct when exercised from some point in the posterior cranial fossa; hence a second reason for the intensity of the symptom in cases of tumor of this locality.

TABLE II.

Seat of Tumor.	Headache.		Vomiting.		Convulsion.		Choked Disk (Bernhardt alone).								
							Amaurosis.		Vision intact.			Total.			
	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.		Cases.		Per cent.	
Cerebral peduncle .	4	40.0	..	...	2	20.0	.....		.....			.....			
Cerebral lobes . .	129	66.0	36	18.5	49	25.0	15 in 124	12.0	12 in 124	10.0	=	27 in 124		21.0	
Basal ganglia . .	19	46.0	8	19.5	7	27.0	.....		2 in 26	8.0	=	2 in 26		7.0	
Cortex	37	50.0	18	24.0	20	28.0	5 in 57	11.5	5 in 57	9.0	=	10 in 57		17.0	
Pons	37	67.0	15	27.0	2	5.0	4 in 30	13.0	2 in 30	7.0	=	6 in 30		20.0	
Medulla	16	60.0	12	40.0	2	6.0	.....		2 in 21	9.0	=	.....		9.0	
Cerebellum . . .	150	83.0	75	38.5	18	9.0	18 in 90	20.0	13 in 90	14.0	=	31 in 90		34.0	
Corp. quad. . . .	9	69.0	8	61.0	1	8.0	5 in 11	45.0	1 in 11	10.0	=	6 in 11		54.0	
Total	423 in 568 cases = 74 per cent.		172 in 568 cases = 30 per cent.		91 in 568 cases = 16 per cent.		82 in 362 cases = 22 per cent.								

Epileptiform convulsions constitute a fourth diffused symptom, which is very characteristic of tumors of the brain. Their causal relations to increased intracranial pressure has been strikingly shown by Leyden's experiments. In these, pressure was directly applied to the brain of animals previously trepanned for the purpose. Convulsions occurred as soon as the pressure had risen to 130 mm. of mercury. Pressure, however, is only one of the mechanisms by which convulsions may be excited. Kussmaul's experiments, made many years ago, showed that sudden anæmia of the brain, such as might be induced by copious hemorrhage, was invariably followed by convulsions. The predominance of convulsions in cases of brain tumor, according to the locality occupied by the latter, does not follow the law which is applicable to symptoms traceable to increased pressure, for convulsions occur oftenest in cases of tumor of the cortex and cerebral lobes, presumably of the portions of the centrum ovale which lie immediately beneath the cortex. General convulsions, therefore, like local spasms, are rendered imminent by direct excitation of the motor tracts. Curiously enough, convulsions are almost excluded from the symptomatology of the pons, though this re-

gion, which is traversed in every direction by motor tracts, probably contains the convulsing centre. But apparently the properties of the centre become abolished before they can be effectually irritated. This absence of convulsions, when certain positive signs are present at the same time, is of real value in localizing a tumor in the pons.

As the convulsion is not proportioned to the locality of greatest tension, so it stands in no relation to the time at which tension is greatest. It occurs as an initial symptom, or during the active period of the disease; but it usually disappears, with other irritative symptoms, in some other manner toward the close, when intracranial pressure is at its maximum. Sudden variations in such pressure, caused by fluctuations of the circulation, seem to be the essential proximate cause of the convulsions of brain tumors. The form of the convulsion does not differ from that observed in idiopathic epilepsy, and the diagnosis between tumor and epilepsy is often difficult. It can only be made by means of the concomitant symptoms.

Apoplectiform attacks occur with brain tumors, and may, though rarely, be the first symptom and followed by paralysis or paresis. It is extremely difficult, then, to distinguish the case from one of ordinary cerebral hemorrhage. Hemorrhage into or around the tumor is a frequent cause of apoplexy, and thus may first reveal the existence of a tumor hitherto latent; or it may occur incidentally among phenomena already well defined and recognized. Finally, the apoplectic attack may usher in the terminal period; the patient never completely recovering, but passing into a soporose condition and finally into coma. The apoplectic ictus is not invariably associated with hemorrhage;

it may be due to sudden alterations of intracranial pressure, by which the functions of brain-tissue are temporarily suspended, as after concussion.

Psychic Changes.—The earliest is usually an extreme irritability, which contrasts with the lachrymose emotionality characteristic of softening of the brain. Occasionally this culminates in attacks of maniacal excitement; oftener, however, the patient suffers from melancholic depression, and gradually becomes more and more apathetic and taciturn. This taciturnity, which is a diffused symptom, must be distinguished from true aphasia. As in all mental disturbances, the memory fails. Dementia may precede death for some time, especially if epileptic convulsions have been severe.

CASE VII., by Mills: Attacks of mania at intervals. Tumor occupies occipital and postero-parietal gyri.

CASE, by Hunt: Speech mumbling, thick, no wrong words, mental confusion, drowsiness, loss of memory. Tumor occupies angular and supramarginal gyri.

When tumor complicates a diffused meningo-encephalitis, the mental symptoms are attributable rather to that.

CASE, by Magnan, (*Brain*, 1879): Angiolithic sarcoma (psammoma), reducing ascending parietal gyrus to one third its volume, associated with diffuse meningo-encephalitis; epileptiform convulsions for eight years; intellectual faculties impaired; loss of memory and moral sense; mania, dementia.

The following case is remarkable for the short duration of the symptoms:

CASE, by Bristowe (*Brain*, 1884): First symptoms fourteen weeks before death. Intelligence early impaired, after headache and right hemiparesis. With progress of paresis patient became stupid, drowsy, finally comatose. Tumor in anterior part of corpus callosum.

TABLE III.

Seat of tumor.	Intelligence Disturbed.										Intelligence Normal.	
	Mental depression, apathy, loss of memory, imbecility.		Hallucinations.		Delirium or mania.		Drowsiness or stupor.		Total.			
	Cases.	P. cent.	Cases.	P. cent.	Cases.	P. cent.	Cases.	P. cent.	Cases.	P. cent.	Cases.	P. cent.
Medulla, 29 cases . .	6	20.0	2	7.0	2	7.0	1	3.5	11	38.0	18	62.0
Cerebellum, 162 cases .	48	29.0	1	...	6	3.0	9	5.0	64	39.0	98	60.0
Cortex, 57 cases . . .	..	...	..	...	..	...	..	...	28	49.0	..	...
Pons, 56 cases	27	48.0	1	...	..	...	1	...	29	51.5	27	48.5
Basal ganglia, 40 cases .	18	45.0	1	...	2	...	..	...	21	52.0	19	47.0
Lobes, 192 cases . . .	90	47.0	9	4.0	11	5.5	5	2.5	115	60.0	77	40.0
Occipital	1	...	..	...	..	...	..	...	..	...	1	...
Frontal	6	10.0	..	...	..	...	..	...	6	10.0	11	19.0
Parietal	9	15.0	1	...	2	...	7	12.0	19	33.0	17	29.5
Temporal	2	3.0	..	...	..	...	..	...	..	...	..	...
Corp. quad., 13 cases .	4	30.0	..	...	2	15.0	4	30.0	10	77.0	3	23.0

It is extremely noticeable from Table III. that the liability to perversion of intelligence is not at its maximum when the tumor is seated at the cortex, nor when a cortical tumor is in the frontal lobes. Cortical tumors stand third from the bottom of the scale, in this respect; the highest place is occupied by tumors of the corpora quadrigemina, seventy-seven per cent. A relative infrequence of mental disturbances is observed in tumors of the medulla and pons. On the other hand, the high percentage of such disturbances in tumors of the centrum ovale may probably be, at least in part, attributed to their influence upon the cortex. To such influence must, in last analysis, all psychic perversions be ascribed; and the high proportion of cases in which these are present with tumor in any locality of the brain, is explained by the extreme sensitiveness of the cortical substance to disturbance of the intracranial pressure from whatever point diffused. Psychic symptoms, of one kind or another, are seen to be extremely frequent in tumors of the brain, being present in about half the

cases. Their presence, therefore, materially aids in establishing the diagnosis.

Choked Disk.—This symptom, when present, is more nearly pathognomonic, than any other, of cerebral tumor. The phenomenon of the choked disk has been regarded as the expression of two different morbid processes—the one an inflammation of the optic nerve, the other a mechanical obstruction to its circulation. In either case, to the ophthalmoscope the papilla appears engorged, tumefied, nebulous, irregular, and with ill-defined edges; a species of cloud covers both the centre and the circumference, rendering the whole surface opaque. The arteries are diminished in calibre; the veins appear interrupted at various points. In one form of choked disk, probably the inflammatory, the capillaries seem increased in size; in the other, effaced.

According to the celebrated doctrine of von Graefe, these appearances are always due to an obstruction offered to the venous circulation of the optic nerve, from mediate or immediate pressure exercised upon the sinus cavernosus. Hence a serous transudation from the veins, rendering the papilla swollen and œdematous. It has been shown, however, that the free inosculation of the ophthalmic vein with the angular branch of the facial suffices to avert complete venous obstruction, even when the circulation in the cavernous sinus has been retarded. Further, a free communication has been demonstrated between the intervaginal lymphatic space of the optic nerve and the subarachnoid space of the brain. It has been shown that a rise of intracranial pressure suffices to force cerebro-spinal fluid into the intervaginal space of the

nerve, thus causing compression of its central vessels, local obstruction, and swelling from transudation, apart from venous obstructions.

Choked disk sometimes appears in cases in which the tumor is so small that much increase of intracranial pressure seems doubtful. It is then more probably due to inflammation of the optic nerve, first propagated from irritated brain-tissue to the central terminations of the nerve—neuritis from diffused cerebritis (Mackenzie), or excited by direct pressure upon the optic tract. The latter may be effected by

TABLE IV.

CASES OF DIFFUSED SYMPTOMS ALONE.

Seat.	Headache.	Headache and vomiting.	Headache and convulsions.	Headache and choked disk.	Headache, convulsions, and vomiting.	Headache, convulsions, and choked disk.	Headache, convulsions, vomiting, and choked disk.	Headache, vomiting, and choked disk.	Vomiting.	Convulsions.	Convulsions and choked disk.	Psychic alteration.	Total.
Cortex, 57 cases . .	4	3	1	..	..	1	..	..	..	1	1	In 7 of these.	11 = 19 per cent.
Lobes, 124 cases . .	5	3	8	2	4	5	..	3	2	4	1	In 22 of these.	37 = 29 per cent.
Basal ganglia, 26 cases	2	1	..	..	..	..	..	..	..	2	..	1 alone, 1 besides.	6 = 23 per cent.
Cerebellum, 90 cases .	2	6	1	..	3	..	..	4	..	1	..	In 4 of these.	17 = 18.5 per cent.
Total	13	13	10	2	7	6	..	7	2	8	2	35	71 = 23.8 per cent.

tumors of the corpora quadrigemina, of the cerebral peduncles, or of the interpeduncular space. Table II. shows that the percentage of cases of choked disk, in cases of intracranial tumor, is greater in the locality of the corpora quadrigemina than in any other. But as shown by Table X., it is only present in twenty-two per cent. of all cases hitherto observed; thus is less frequent than any of the diffused symptoms except convulsion.

Choked disk is found far more frequently (54 per cent.) in cases in which the tumor involves the corpora quadrigemina, than in those in which it involves any other part of the brain. The smallest percentage is in the class of cases in which the basal ganglia are the seat of the tumor. When there is direct pressure on the optic tract, the papilla sometimes atrophies without passing through any stage of choked disk. Until atrophy sets in, vision is not necessarily impaired. Thus, out of a total of 82 cases of choked disk, vision remained intact in 37, or 45 per cent. In a great many cases no ophthalmoscopic examination is made, unless vision is impaired, and this explains why such examination is lacking to the history in 232 out of 485 observations analyzed by Bernhardt (47.8 per cent.). In many of these negative cases it is very possible that choked disk really existed, so that the real proportion of this lesion in brain-tumor cannot be considered as known.

In a certain number of cases cerebral tumor manifests itself exclusively by one or more of the foregoing "diffuse" symptoms. Among Bernhardt's cases of tumors of the cortex, centrum ovale, cerebellum, and basal ganglia, this limitation may be found seventy-one times out of a total of 297 cases, or 23.8 per cent.

The existence of mental symptoms in a large proportion of these cases (forty-nine per cent. of them) is the circumstance that might, perhaps, most surely guide in the diagnosis, otherwise so difficult.

The proximate consequences of increased intracranial pressure, and which are the immediate cause of the diffused symptoms, have been differently interpreted. It was long maintained that the brain sub-

stance was nearly as incompressible as water. Room, therefore, could only be made within the cranium for a neoplasm by proportionate expulsion of blood and lymph, and by atrophy of the brain-tissues in the immediate vicinity of the tumor. Adamkiewicz's experiments have shown, however, that the nerve-tissue surrounding the tumor is compressed, *i. e.*, its solid molecules are approximated, and the fluid, normally interposed between them, is, to a greater or less extent, expelled. For, when a piece of laminaria was inserted under the skull of an animal previously trepanned for the purpose, and was allowed to swell, thus rapidly encroaching upon the intracranial space, microscopic examination of the tissue in which the foreign body was embedded, revealed the fact that all the nerve-elements of this tissue were closely crowded together, thus apparently multiplied in a given space. The zone adjacent to this was intensely vascularized from dilatation and new development of capillaries, and, in addition, it was hypertrophied from proliferation of connective tissue.

In the experiment, the swelling of the laminaria was much more rapid than is the growth of any tumor, and the condensation and nutritive irritation or tissue were, therefore, exaggerated. To a greater of less extent, however, both these lesions must always be produced by the presence of a foreign body within the cavity of the cranium. Only when the tumor grows very slowly are they absent, or reduced to such a minimum as to occasion no symptoms, either diffused or focal.

The occurrence in 23 per cent. of the cases, of diffused without focal symptoms, indicates that the centres of origin of nerve tracts have remained unaf-

fected, though the vomiting and convulsive centres and the nervous filaments of the dura mater have been irritated; that lymph has been forced into the sheath of the optic nerve, or that a descending neuritis has been excited by propagation from the zone of cerebritis surrounding the tumor,[1] and that the delicate psychic mechanisms of the cerebral cortex have been irreparably jarred and are out of working order.

This immunity of motor, sensory, or special-sense nerve-functions, is usually due to the localization of the tumor in a "latent" portion of the brain; but it is also sometimes observed in cases in which the tumor occupies a (presumable) focus of nerve-origin. Thus, although there can be no doubt that the ultimate origin of the motor nerves contained in the pyramidal tracts is in the central gyri and paracentral lobule, tumors seated in these localities have sometimes been observed entirely unaccompanied by motor symptoms, either irritative or paralytic. Two such cases are contained among the eleven of the table. In one of these there were forty hydatid cysts in the brain, a form of neoplasm very frequently latent. In the second case, however, there was a most extensive sarcoma occupying the lower half of the anterior central gyrus, posterior half of third frontal gyrus, and under half of insula.

Two explanations are offered for these cases. First, that the elements of the neoplasm have insinuated themselves so gradually between those of the nerve-tissue, or have displaced them with so little injury that the functions of this tissue have not suffered. This explanation applies to cases in which, instead of

[1] Demonstrated by Adamkiewicz (see *ut supra*).

the cells of a nucleus of origin, the fibres of a nerve-tract have been displaced, as in some extraordinary cases on record in which a tumor has occupied nearly the entire pons, yet has occasioned no motor symptoms. The second explanation applies only to cortical centres. According to Exner, the different mechanisms of the cortex, though specially concentrated at certain localities, exert their influence somewhat beyond these limits, though with constantly diminishing intensity and effectiveness. Hence it is occasionally possible, though the main centre be destroyed, that its action may be supplemented by that of others habitually subordinate.

In more than three fourths of the cases of brain-tumor, in addition to the diffused symptoms hitherto described, the patient suffers from perversions or abolition of one or more cerebral functions other than psychic ones. These are known as the focal symptoms.

Focal Symptoms.—They consist of the perversion or abolition of mobility or sensibility in one or more cranial nerves or spinal nerve-tracts, or in similar alterations of one or more of the special senses. Among the latter, however, is to be excepted the impairment of vision directly traceable to choked disk or optic neuritis. An intense interest has recently attached to these symptoms as a means of unravelling the physiological problems of the localization of brain functions. For this purpose, however, the study of tumors is much less valuable than that of other brain lesions, such as, for example, localized softening; for their limits are irregular, and their effects, through transmission of pressure, often diffuse themselves in structural or functional changes far beyond these vis-

ible limits. For clinical purposes, therefore, it is necessary to ascertain, first, what symptoms are generated by lesions really limited to certain localities; second, to what extent the complication of these by others, diffused or symptomatic of different localities, may aid us in diagnosing the existence of tumor as distinguished from other focal disease.

Focal symptoms are always unilateral at the beginning—a most useful criterion in distinguishing tumor (as well as other localized lesions) from a diffused disease of the brain. The appearance of symptoms on the opposite side of the body from that on which they first began, indicates an extension of the growth across the median line. This, for obvious reasons, most frequently occurs at the narrowest regions of the encephalon, the pons and (though less frequently) the medulla. It is, however, also seen in tumors of the corpus callosum, but the second hemiparalysis is much slighter than the first.

Case (Bristowe, *Brain*, October, 1884) : Illness twelve weeks. Left hemiplegia, gradually extending to right side ; then general paralysis, principally on the left side, ten days before death. Progressive drowsiness or stupidity, aphasia. Sarcoma occupied anterior two-thirds of fornix and corpus collosum, extending into the centrum ovale in both hemispheres, but principally in the right.

In addition to these symptoms involving purely cerebral functions, the functions of respiration and circulation are sometimes modified from the direct or indirect morbid influence exercised upon the medullary centres.

Lesions of Motility.—These are by far the most numerous, the most varied in character and in combination of all the focal symptoms of brain tumor. They belong to three different classes: First, irrita-

tive, including tremors, choreiform movements, and local spasms[1]; second, ataxic, implying inco-ordination among functionally combined movements; third, paralytic, consisting in the partial or complete abolition of motive power.

Irritative Lesions of Mobility.—A fine tremor or a clonic spasm, incessant or periodically repeated, is often seen, either in muscles which have already become paralyzed, or in those which become paralyzed at a later date.

Case (Berger, *Arch. der Heilkunde*, XIX. Jahr.): Woman, aged forty-eight. During a year, about every eight days, an attack of clonic spasms in the right arm, then paralysis of the same arm, followed by paresis of the buccal branches of the right facial; clonic spasms persist after paralysis sets in; death a week later. Tumor in left anterior central gyrus, compressing the posterior and second frontal gyri.

Case (Berkley, *Med. News*, 1882): Patient with spasm of the left angle of the mouth for two and a half years. Sudden death from cardiac disease. Calcareous nodule three sixteenths of an inch in diameter on the right ascending frontal convolution, one and a half inch above the fissure of Sylvius; the locality corresponds to Ferrier's centre for the zygomatic muscles.

Tremors and localized spasms are valuable diagnostic symptoms; for, first, they are more frequent with tumors than with other localized brain lesions; second, they are more frequent in the cortex; and, third, they are especially frequent in the motor zones. All these circumstances are demonstrated by the following tables. The first is compiled from Exner's collection of 164 cases, exclusively of cortical lesions.

Tumors of cortex (44 cases): Spasm, 3 = 6.5 per cent.; spasm and paralysis, 14 = 31 per cent.; paraly-

[1] The general epileptiform convulsion being a diffuse symptom.

sis, 14 = 31 per cent.; no motor symptoms, 13 = 29 per cent. Total spasm, 17 = 38 per cent.

Other lesions of cortex (100 cases): Spasm, 1 = 0.9 per cent.; spasm and paralysis, 13 = 11.5 per cent.; paralysis, 62 = 56 per cent.; no motor symptoms, 36 = 32 per cent. Total spasm, 14 = 12 per cent.

Thus, in more than one third of all cases of brain tumor, localized spasms or contractures exist at some period of the disease. When present they indicate a greater probability of localization in the cortex than in any other part of the brain; and after that, in the region of the corpus striatum and thalamus opticus. In the table the highest percentage falls to tumors of the peduncle; but this fact is offset by the great rarity of tumors in this region.

TABLE V.

PROPORTION OF CASES OF SPASM WITH TUMORS.

Seat.	No. of cases.	Spasm.	Spasms and paralysis.	Paralysis.	Total spasm.	Percentage.	No motor symptoms (except ataxic).
Cortex—							
Central gyri	39	4	24	10	28	71	2
Parietal lobe (motor)	11	...	7	1	7	63	3
Total motor zone	50	4	31	11	35	70	5
Frontal gyri	14	2	1	4	3	21	7
Other latent parts	13	1	2	2	3	23	8
Total cortex	77	7	34	17	41	53	Perct. 20 = 25.0
Centrum ovale	124	12	22	45	34	27	45 = 36.0
Basal ganglia	41	9	10	14	19	46	8 = 19.0
Peduncle	10	1	5	3	6	60	2 = 20.0
Corp. quad.	13	1	3	2	4	30	7 = 53.0
Pons	56	3	7	38	10	17	8 = 14.0
Cerebellum	165	20	12	26	32	19	107 = 64.5
Medulla	30	8	2	15	10	33	5 = 16.0
Total	516	...	...	...	156	30	202 = 37.5

It is evident that spasmodic contractions of muscles may be caused by irritation, either of the nerve-

elements of a motor-centre, or of the fibres of a motor-tract descending from it, but that the first condition is more favorable. Tumors of the pons and medulla are rarely accompanied by spasm ; it seems that the liability to irritation increases higher up in the tract, and also when the latter is more incompletely invaded.

Paralyses of Motility.—These are especially characterized, as a rule, by their gradual development, a circumstance which is most useful in distinguishing brain tumors from hemorrhage. It does not, however, serve to differentiate tumors from softening ; for in the latter the paralysis is also gradually developed.

To a certain extent the paralyses of tumors share the peculiarities of those caused by other lesions of the same locality. As already stated, however, in the case of tumors, the paralyses are rarely purely typical throughout the whole course of the disease, because they constantly tend to encroach upon other regions than that in which they originated, and because their influence, by transmitted pressure and nutritive irritation, is apt at all times to diffuse itself considerably beyond the region which they visibly occupy. A paralysis which may seem at a given stage to be entirely atypical, may, however, exhibit in the history of its development peculiarities which point out the true nature of the disease. The paralysis has been preceded by a slowly progressing paresis, or by tremor or spasm in the affected muscles, or has existed in one set of muscles, or in one limb, or in one or more cranial nerves; or there has been a combination of paralyses of such nerves with others of the extremities before the disease reached its complete evolution. Or, further, the very first appearance of a paralysis may

have been preceded by one or more diffused symptoms; or it may have been ushered in by an epileptiform convulsion or an apoplectic attack, remarkable for its brevity and incompleteness. Or a paralysis may declare itself at once, in a fully developed form, but isolated, as in one facial nerve, and after prolonged headache, attacks of vomiting, and change of mind or character of the patient. The typical characteristics of the paralyses, according to locality, are as follows:

Cortex.—The paralysis, at the outset at least, is "dissociated," monoplegic. One arm or one side of the face is affected, or the two together are affected on the same side. It is extremely rare that paralysis begins in the leg; but this extremity often becomes involved later, and then the patient suffers from a complete hemiplegia, difficult to distinguish at first from the common hemiplegia due to hemorrhage into the internal capsule. It is very rare, however, that the paralyzed limbs become rigid. It is with tumors in this region that clonic spasms are most frequent either before or during the paralysis. Symptoms of tumors of different regions of the cortex follow, approximately, the rules which have been laid down for other lesions, according as they occupy the "latent" or the motor zones. The latent regions are those parts of the brain in which, with rare exceptions, lesions produce no motor symptoms. The motor zones are those whose lesions are always followed by spasm or paralysis, except in very few cases, in which the absence of symptoms is explained by the extremely slow growth of the tumor which allows nerve-tissue to accommodate itself to increased pressure. When an "absolute field" exists it will be found that in all cases in which motor symptoms are absent this

field is entirely free from lesion. In the regions adjacent to these, lesions sometimes do and sometimes do not produce symptoms. This fact, as already stated, has been explained in two ways—by the theory of transmitted pressure, and by the theory of a "relative field," which contains motor mechanisms of less degree of intensity and concentration than those belonging to the "absolute field." The absolute motor zones are :

First, for the upper extremity, the anterior central convolutions, especially the lower two-thirds, the upper half of the posterior central convolution, the paracentral lobule, and, in the left hemisphere, the greater part of the superior parietal lobe, and possibly a few points on the occipital.

Second, for the lower extremity, it is the upper third of both central convolutions and the paracentral lobule, and in the left hemisphere again the greater part of the superior parietal lobe. This "absolute field" is, according to Exner, surrounded by a relative field which occupies the posterior half of the superior frontal gyrus, almost the entire convex surface of the other two frontal gyri, both parietal lobes, and the upper part of the occipital lobe. This field belongs to both extremities.

Third, there is no absolute field for either facial muscles or tongue, the mechanisms for both seeming to be diffused over the greatest part of the hemispheres. But the seat of greatest concentration for the facial nerve exists in the lower half of the anterior central gyrus, and the lower third of the posterior central, while a relative field extends over the posterior half of both lower frontal gyri and the anterior part of the supramarginal gyrus. The principal

centre for the hypoglossal nerve is the lower part of the anterior central gyrus and adjacent part of the middle and inferior frontal gyri.

Fourth, no definite cortical field has been outlined for either the motor oculi nerve or the trigeminus. In regard to the first, however, it seems certain that all the branches of both nerves are influenced by the centres of a single hemisphere.

The zone for common sensibility coincides with the motor zone as above defined.

Fifth, the zones latent in regard to motor or sensory symptoms include all the frontal lobes, the temporo-sphenoidal lobes, the parietal lobe of the right hemisphere, and the occipital lobes. Lesions of these lobes may remain absolutely latent, and did so in 13 of Exner's 44 cases of tumors; that is, in 29 per cent. But even when unattended by paralysis or spasm lesions of these latent zones are liable to be followed by such disorders of speech, of vision, or of hearing as lead to the localization, within their boundaries, of the centres for these important functions.[1] In Table V. it will be seen that there were 9 cases of paralysis, with or without spasm, occasioned by tumors in the non-motor regions; the percentage of paralysis to whole number of such tumors being 37. Out of the whole number of cases of paralysis from 77 tumors of the cortex (51 cases), the percentage belonging to tumors of non-motor regions was 17.5 per cent.; that of those belonging to motor regions (42 cases) was 54 per cent. of the whole, and 84 per

[1] The wide diffusion of the mechanisms for the motor-oculi nerve and the facial, which render their paralyses of little value in regional diagnosis, is probably correlated to the complex relations of these two nerves to the mechanisms of psychic existence, and their functions in the innumerable shades of facial expression.

cent. of the tumors of that region; while, finally, the probability that a tumor situated in the cortex would occasion some form or degree of paralysis is indicated by the relation of 51 to 77, or 66 per cent.

Centrum Ovale.—A much larger percentage of tumors remain latent in this region than in the cortex, as, for example, 36 per cent., instead of 25 per cent. (see Table V.). The absence of symptoms is to be expected when the tumor neither occupies nor affects bundles of fibres coming from the motor regions of the cortex. In the following table, Ladame's and Bernhardt's cases are combined, and show to what extent tumors situated in non-motor regions may yet inhibit the mechanisms of the motor regions:

TABLE VI.

PARALYSIS WITH TUMORS OF CENTRUM OVALE.

MOTOR REGIONS.			NON-MOTOR REGIONS.		
Seat.	Paralysis.	No. Paralysis.	Seat.	Paralysis.	No. Paralysis.
Pars centralis anterior and posterior (Petres) . . .	61	16	Pars frontalis . .	37	37
			Pars occipitalis .	10	23
			Temporal lobe .	2	3
			Other parts . .	..	7
Total	61	16	Total	49	70

The paralyses which are associated with tumors of this region present nearly the same characteristics as do those associated with tumors of the cortex if they are near the surface; that is, they are liable to be monoplegic; but they resemble those of tumors of the internal capsule if they approach the basal ganglion, in which case they may become completely hemiphlegic, and may be followed by rigidity. Usually a long period of paresis precedes that of complete paralysis.

The percentage of cases of paralysis in tumors of the centrum ovale, whether calculated from the smaller number of cases in Table VI., or from the larger number in Table V., is about the same, viz.: 54 in the first case, 56 in the second.

Basal Ganglia.—Tumors of the corpora striata, optic thalami, and lenticular nuclei occasion hemiplegias, which often differ from those of hemorrhage in the same region, exclusively by their gradual rate of development. The paralysis is, however, sometimes monoplegic; thus, out of 41 cases it was confined to the facial nerve four times, to the arm once, to the arm and facial once, and to the leg once. It is extremely remarkable that large tumors may exist in this region without causing any symptoms whatever. This is the rule for tumors limited to the thalamus or to the lenticular nucleus. Acute lesions, such as hemorrhage in the latter ganglion, cause temporary hemiplegic symptoms; but these subside, probably because the function of the destroyed tissue is supplemented by that of other motor centres. But such temporary paralyses are not seen with chronic lesions, as, for example, tumors, unless they are complicated by an accidental hemorrhage.

But tumors limited to the corpus striatum will certainly cause paralysis if they involve the anterior two-thirds traversed by the motor tract of the internal capsule. It is injury to this tract which determines the phenomenon of "late rigidity"; a phenomenon depending on the descending degeneration which reaches the spinal cord, and which, though so commonly seen after cerebral hemorrhage, is not peculiar to that lesion, but only to the locality which it most frequently occupies.

If a tumor involve the posterior third of the internal capsule, whose fibres pass between the corpus striatum and the thalamus, it tends to destroy the sensory fibres which pass in this locality (Charcot, Veysière), and to cause a hemianæsthesia in addition to the motor paralysis. This complication is, therefore of great use in establishing the diagnosis of tumors of this region, which, from their encroaching tendencies, are so liable to involve all parts of the internal capsule. It is possible that a transmitted irritation to sensory fibres has something to do with the high percentage of spasms observed in tumors of the basal ganglia (46 per cent., see Table V.). There were 24 cases of paralysis, with and without spasm, which is 58 per cent. of the whole number.

Peduncle.—As might be expected, tumors of this region cause hemiplegic paralysis in almost all cases (80 per cent.). Together with the extremities, the facial nerve and also the hypoglossus are usually involved. The most characteristic circumstance, however, is the paralysis of the motor-oculi nerve by direct pressure upon its trunk as it emerges in the interpeduncular space. The paralysis is on the same side as the tumor; that is, on the side opposite to the hemiplegia. The paralysis is usually total, in which case there will be unilateral dilatation of the pupil, ptosis from paralysis of the levator palpebræ muscle, and divergent strabismus from paralysis of the internal rectus. In other cases, one or more of these symptoms may exist alone. As the tumor grows larger, it sometimes crosses the interpeduncular space, and compresses the nerve on the opposite side. This important symptom existed in seven out of the ten cases of Ladame and Bernhardt. It is not, however,

absolutely pathognomonic of lesions of the peduncle; for it results, with exactly the same forms, from every tumor of the interpeduncular space; thus, from those springing from the base of the cranium.

Corpora Quadrigemina.—Tumors of these bodies lie outside of the direct cerebro-spinal motor tracts, and thus produce much less definite motor symptoms. Some degree of paralysis existed in 5 out of 13 cases; in 1, paresis of the arm and facial nerve; in 2, a unilateral facial paresis; in 1, paresis of one leg; and in 1, paresis of one half of the body.

On the other hand the motor-oculi nerve seems to be paralyzed as often as in the case of tumors of the peduncles; a fact which might be expected from the proximity of the corpora quadrigemina to the nuclei of the nerves which lie immediately below them. In 14 cases divergent strabismus existed in 8 (five cases of Bernhardt, three related by Nothnagel). In one other case the abducens was paralyzed, so that internal strabismus existed (Gowers, *Lancet*, 1879).

Cerebellum.—Absence of true motor paralysis, taken together with impairment of the power of equilibration, is highly characteristic of tumors of the cerebellum. Out of a total of 165 cases, only 38 showed any kind of paralysis (23 per cent.). This is almost the proportion in which the symptom is absent in tumors of the cerebral cortex. Excluding the cerebellum and corpora quadrigemina, the probabilities of paralysis with brain tumor are expressed by the percentage 89, while for the cerebellum and corpora quadrigemina alone the percentage is only 24.

Tumors of either lateral lobe of the cerebellum cause of themselves no motor symptoms, even ataxic, and may be completely latent. Out of the 38 cases

of paralysis the tumor occupied the middle lobe of the cerebellum in 4; in 5, one of the peduncles; in 15, though mainly situated in a lateral lobe, it extended into the middle lobe, or else compressed the pons or medulla.

The facial nerve may be affected either by an isolated, by an alternating, or by a hemiplegic paralysis, in the rare cases in which hemiplegia occurs. The lesion is never really of cerebellar origin, but always secondary to encroachment upon the pons or medulla.

Pons Varolii.—In this locality tumors produce the most extensive and also the most complex combinations of paralyses. They are occasionally paraplegic, and not infrequently they become, little by little, generalized throughout the four limbs. This creeping generalization is highly characteristic of tumors of the pons. On the other hand, only cranial nerves may be affected.

Case (Wernicke, *Archiv. f. Psychiat.*, Bd. vii.): Patient aged fifty-eight years. In July, headache, diplopia, difficulty in opening and shutting mouth. By end of August, paralysis of left facial nerve, including upper branches; rigidity of left masseter; eyes persistently deviated toward the right; diminished sensibility of face and head on the *right* side; that is, on the side opposite to the facial paralysis. Death occurred in October without further motor affection. Section discovered a tumor on the floor of the fourth ventricle, on the left side of the middle line. Associate nucleus of facial and abducens completely destroyed; left facial nerve nucleus, as also part of the fibres of the right trigeminus destroyed.

After the frequent generalization of the paralysis, the remarkable symptoms of pontine tumors are: 1. The coexistence of hemiplegic paralysis of the extremities with paralysis of one or more cranial nerves

on the opposite side of the body; alternate paralyses. 2. The occurrence of a *persistent* conjugate deviation of the eyes, thus distinguished from the same symptom in lesions of the hemispheres, where it is always transitory. To these positive symptoms may be added an important negative characteristic, namely, the nearly complete absence of local irritative symptoms, and, to an even more marked degree, of general convulsions. The alternate paralyses are produced by tumors in the lower part of the pons, which injure the nerve-nucleus or compress the nerve-trunk on the side on which they are situated, and injure the general motor tracts of the limbs previous to their decussation, so that the resultant hemiplegia follows the usual law for cerebral paralysis, and appears on the side of the body opposite to the lesion. When the tumor occupies the upper segment of the pons anterior to the cerebral peduncles, the facial paralysis will be on the same side as the limb, since it depends, not on a lesion of the nucleus, or nerve-trunk, but on one involving the central fibres after their decussation.

In the most typical cases all the branches of the facial are paralyzed, including those innervating the orbicularis palpebræ. The eye cannot be closed, and the patient presents the appearance of Bell's paralysis. The electric excitability of the nerve may then be diminished. However, neither of these last conditions is invariable, even when the paralysis is alternate.

Double facial paralysis is extremely rare. It is lesions of the pons which have furnished the explanation of the remarkable phenomenon—conjugate deviation of the eyes—which for a long time puzzled pathologists. This deviation implies paralysis of the

abducens nerve of one side, supplying the external rectus, and coincident paralysis of a branch of the motor-oculi nerve supplying the internal rectus on the opposite side. The apparent remoteness from each other of the nuclei of origin of these two nerves rendered this phenomenon extremely difficult to understand, until the discovery was made, in the pons, of a common nucleus, which unites fibres of the abducens with fibres from the lower nucleus of the motor-oculi on the opposite side. Destructive lesions of this associate nucleus are followed by a permanent conjugate deviation, as in the case (Wernicke) above quoted. It becomes evident that the transitory deviations of the eye, frequently seen immediately after an attack of hemorrhage into any part of the brain, are due to a remote shock propagated to this same nucleus.

The abducens nerve is not infrequently paralyzed alone, causing a converging strabismus of the affected eye.

Isolated paralysis of the motor-oculi nerve is much more rare, and is seen only when the tumor or its influence extends above the pons into the cerebral peduncles, or above them to the nerve nuclei. Ptosis, from isolated paralysis of the levator palpebral branch, has sometimes been observed alone, and, so far, in cases of tumors, but not in those of any other lesion. This symptom would, therefore, be useful in differential diagnosis.

Paralysis of the hypoglossus is not rare. It is indicated by an impairment of the voluntary movements of the tongue and by disturbance of speech, anarthria. This paralysis alternates with that of the extremities. It is distinguished from progressive bulbar paralysis by absence of atrophy of the tongue.

The motor branch of the trigeminus is sometimes paralyzed, more often irritated, causing, in the latter case, spasmodic trismus, or clonic convulsions of the muscles of mastication.

Difficult deglutition is also sometimes present, but does not seem to be attributable to paralysis of the pharynx muscles, but rather to be a secondary consequence of paralysis of the tongue and of certain muscles innervated by the facial nerve, the styloglossus, digastricus, and stylohyoideus (Nothnagel).

The following table exhibits the various combinations of paralysis which have been observed with tumors of the pons :

TABLE VII.

MOTOR PARALYSES WITH TUMORS OF PONS (56 Cases).

Cranial nerves alone.	Limbs alone.	Combination of limbs and cranial nerves.	No motor symptoms.
3d nerve 2	Hemiplegia . . . 7	*On same side.*	
7th nerve 3	Paraplegia 3	Hemiplegia and 7th nerve 4	
3d and 6th nerves . . 2	Four extremities . 2		
6th and 7th nerves . 3	Arm alone 1	*Alternate paralysis.*	
7th and 12th nerves . 1		Hemiplegia and—	
3d, 7th, and 12th nerves 1		3d nerve 2	
3d, 5th, 7th and 12th nerves 1		6th nerve 3	4
		7th nerve 4	
		3d and 6th nerves . 1	
		3d and 7th nerves . 3	
		6th and 7th nerves . 5	
		3d, 6th, and 7th nerves 1	
		3d, 5th, 7th, and 12th nerves 1	
		3d, 6th, 7th, and 12th nerves 1	
Total 13	Total 13	Total 25	4

The number of cases in which the cranial nerves or those of the limbs were paralyzed independently of each other is, in this collection of cases, exactly equal. The number of cases of combined paralyses is just double that of either of the classes of isolated paralyses. Among the cranial nerves, the liability of the

facial is evidently the greatest. It was affected, alone or in combination, twenty-four times; the abducens sixteen times.

Medulla.—In this region the liability to paralysis again diminishes. Tumors of the medulla are not infrequently confined to the floor of the fourth ventricle, so that the motor tracts and nuclei are both left uninjured. In this case the patient escapes all paralysis; indeed, he often remains with singularly few symptoms for the subject of an organic disease seated so near to vital nerve-centres. Out of 30 cases, 12, or nearly half, remained free from motor symptoms. In one case, so far unique (Erichsen, *Petersb. Med. Zeitschr.*, 1870), a bilateral paralysis of the vocal cords was noted, due to lesion of the accessory nerve.

TABLE VIII.

MOTOR PARALYSES WITH TUMORS OF MEDULLA (30 CASES).

Cranial nerves alone.	Extremities.	Combination.	Negative.
3d nerve 2	Hemiplegia . . . 1	Hemiplegia and 6th nerve 1	
7th nerve 2	3 extremities . . . 1		
3d and 7th nerves . . 1	Paraplegia 2	*Same side.*	
7th and 11th nerves . 1	General 2	Hemiplegia and—	
5th, 6th, and 7th nerves 1		6th nerve (alternating) 1	
		3d, 6th, and 7th nerves 1	
		3d, 7th, and 12th nerves 1	12
		7th and associated 3d and 6th nerves 1	
Total 7	Total 6	Total 5	12

Ataxia.—This third form of motor lesion is principally seen with tumors of the cerebellum and corpora quadrigemina; the latter, possibly from the connection of these bodies with the cerebellum through the superior cerebellar peduncles. In the pons and medulla, the advent of paralysis is often preceded for some time by a staggering or reeling gate "like a

drunkard's." This same symptom is very conspicuous in tumors of the cerebellum, and, when associated with the negative symptoms of absence of motor or sensory paralysis, points very strongly to tumors of this region. For the development of the symptom, however, it is essential that the middle lobe be involved or indirectly affected. Tumors limited to a lateral lobe are characteristically latent.

Forced movements or inclinations of the body or head to one side or the other are sometimes associated with tumor in a lateral peduncle on the corresponding side. A tendency to fall forward or backward has been associated with the situation of the tumor in the anterior or posterior extremity of the upper or lower processus vermiformis (middle lobe).

Lesions of Sensibility.—With the exception of headache, already described as a diffuse symptom, alterations of sensibility are very much less prominent in the symptomatology of tumors than alterations of motility.

It is evident from this table that, in the cortex, the seat of sensibility coincides with the seat of motility. Pain or anæsthesia rarely exists without paralysis, or except in connection with tumors situated in the motor zones. The liability to pain, other than headache, with tumors of the centrum ovale, is very slight (five cases out of one hundred and twenty-four).[1]

It has already been pointed out that tumors of the basal ganglia will cause hemianæsthesia in paralyzed limbs, provided they involve the bundle of fibres which pass in the posterior third of the internal cap-

[1] The percentage of headache, however, was sixty-six, the highest after the cerebellum and rare cases of corpora quadrigemina. The liability to headache, from distension of the dura mater, is constantly seen to bear no proportion to perversions of sensibility due to lesion of sensory tracts or centres.

TABLE IX.

LESIONS OF SENSIBILITY WITH BRAIN TUMORS.

SEAT.	WITH MOTOR PARALYSIS.				WITHOUT MOTOR PARALYSIS.				NEGATIVE.	No. of cases. Total percentage lesions of sensibility.
	Unilateral.		Double.	Trigeminal.	Unilateral.		Double.	Trigeminal.		
	Pain.	Anæsthesia.	Pain.	Pain.	Pain.	Anæsthesia.	Pain.	Anæsthesia.		
Cortex (57 cases):										
Central gyri	6	7	...	...	1	...	...	...	11	14 = 56
Parietal gyri	1	3	...	...	...	1	...	...	12	5 = 29
Frontal gyri	...	...	...	...	1	...	...	...	9	1 = 10
Temporal	...	2	...	...	...	1	...	...	1	3 = 75
Occipital	...	...	...	...	...	...	...	...	1	0 = 0
Entire cortex	8	15	...	...	...	...	...	...	34	23 = 40
Centrum ovale (124 cases)	5	14	...	...	...	...	...	...	105	10 = 15
Basal ganglia (39 cases)	1	5	...	...	...	2	...	...	31	8 = 25
Peduncle (10 cases	1	3	...	...	...	...	...	1	5	5 = 50
Pons (51 cases) Same side, 1 Opp. side, 7	Same side, 1	10	...	2	...	...	...	...	24	27 = 52.5
Medulla (30 cases)	Opp. side, 7	...	2	1	5	...	...	...	16	14 = 46
Cerebellum (167 cases)	4	5	1	1	1	...	...	...	142	25 = 14.5
Corp. quad. (13 cases)	2	7	1	1	5	...	...	...	11	2 = 15
	1	...	...	...	1	...	...	...		
Total (491 cases)	30 = 6 % of all cases.	59 = 12%	... 5 = 1 %	4 = 0.8%	8 = 1.2%	8 = 1.6%	...	1	368	...
	Percentage of lesions of sensibility in all cases equals twenty-five.									

sule, otherwise they will not be attended by lesions of sensibility. The table, therefore, expresses the probabilities of this precise situation, in giving the propor-

tion of cases of pain or anæsthesia as eight out of thirty-nine, or twenty per cent.

The highest percentage is with tumors of the pons, and the next highest, if the few cases of tumors of the peduncles of the cerebrum be excluded, is with those of the medulla. In these places occur pain and anæsthesia in the facial range of the trigeminus, symptoms almost peculiar to such tumors. When similar symptoms are excited by tumors of the cerebellum, it is only because the pons or medulla has been compressed. Trigeminal neuralgia or anæsthesia is, like cramp or paralysis of the masticatory muscles, a most important symptom for helping to localize a tumor in the posterior cranial fossa. It is noticeable (see table,) that trigeminal anæsthesia has hitherto been observed on the side opposite to the paralysis, while anæsthesia of the extremities has nearly always existed on the same side.

The cerebellum and corpora quadrigemina show the same minimum liability to lesions of sensibility as they do to motor paralysis. Their percentage, almost alike for the two cases, is however, not lower than that of the centrum ovale.

Comparison of the latter with the cortex on the one hand, and with the peduncle, pons, and medulla on the other, seems to indicate that sensation is affected either by lesion of terminal nerve-cells (as in the cortex), or of very concentrated bundles of nerve fibres. When these are widely disseminated, as in the centrum ovale, so that a few can only be involved in the lesion, motor power may, nevertheless, suffer extremely, while sensibility remains intact.

Lesions of the Special Senses.—*Vision.*—Disturbances of vision are extremely frequent as symp-

toms of brain tumor, and are of three kinds: First, atrophy of the optic papilla as a consequence of choked disk, and therefore as a remote consequence of increased intracranial pressure; second, deviations of the eyeball or eyelids from isolated or combined paralyses of the nerves supplying the ocular muscles, the third, sixth, and seventh; third, finally, amblyopia or amaurosis, resulting from direct affection of the optic nerve in its course through the cranium, or at its cerebral centres, the mode of development being therefore almost precisely analogous to that of paralysis of any other nervous tract by direct compression. The first two kinds of ocular defect have been sufficiently described; the third comprises two different kinds of lesions, those affecting (by compression) the optic tract or chiasma, and those which affect the optic stations of the posterior extremity of the thalami or at the corpora quadrigemina, or else at the final visual centres of the cortex.

The optic nerve or chiasma is liable to compression from tumors arising from the base of the cranium, or from the hypophysis, and also from tumors of the peduncle; an acute descending optic neuritis, with atrophy of the papilla, is usually excited. When one tract or one side of the chiasma is compressed, hemiopia results, a phenomenon dependent on the semi-decussation of nerve fibres which takes place in the human chiasma. Thus pressure on the right side beyond the chiasma, of such a nature as to injure the fibres of one tract, will abolish vision in the right half of both eyes. A tumor in front of the chiasma may cause temporal hemiopia of both eyes, since it injures fibres coming from the nasal half of both

eyes. There is no way in which a double nasal hemiopia can be produced by tumors at the base of the brain.

Tumors of the thalamus might be expected to affect the sight from lesion of the corpus geniculatum, with its branch to the optic tract. As a matter of fact, however, blindness is not very common from tumors of this locality—only five cases out of twenty-six (nineteen per cent.). Tumors of the corpora quadrigemina, however, have an immensely large proportion of cases. Out of eleven, nine showed either amblyopia or amaurosis, five with and four without choked disk (eighty-one per cent.).

Visual defects from lesions of the cortex are extremely interesting in connection with two physiological problems, viz., the question of a second decussation of optic-nerve fibres in the cerebrum (Charcot), and that of the localization of the mental centre of vision. This centre was placed by Ferrier at the angular gyrus, as an inference from direct experiment upon the brain of monkeys. But Exner, on the authority of four cases of lesion reaching to the cortex, of which two were tumors, places the visual centre in the first and second occipital gyri—the cuneus and adjacent part of the lobulus quadratus.

Case (Gowers, *Lancet*, 1879) : Visual hallucinations of a peculiar nature, associated with some degree of amblyopia, affecting both eyes, but more markedly the left. Tumor occupying first and second occipital gyri, posterior half of superior and inferior parietal lobes, the cuneus, and a part of the lobulus quadratus.

Case (Jastrowitz, *Centralbl. für prakt. Augenheilkunde*, vol. i., 1877) : Paralysis of both right extremities and facialis ; aphasia, with agraphia ; hemianopsia dextra. Tumor of the left occipital lobe, principally in the occipital gyri and the præcuneus.

Case (Pooley, *Arch. f. Augen. und Ohrenheilk.*, Bd. vi.):

Together with various characteristic symptoms of brain tumor in a syphilitic man, extensive binocular hemianopsia. Tumor in posterior lobe of left hemisphere, surrounded by extensive zone of softening. Left thalamus completely softened.

A tumor of one hemisphere may thus cause double hemiopia, a single or double amblyopia or amaurosis, and visual hallucinations of various kinds. The double hemiopia from cerebral lesion, has been interpreted as a proof that, arrived at the cerebral hemispheres, optic fibres which had decussated in the chiasma, recrossed to the opposite hemisphere, thus finally arriving at the same side as the retina, from which they started. Hemiopia is habitually unaccompanied by choked disk. It is indeed rare as a symptom of tumor, and has been principally studied in connection with other lesions.[1]

Six cases of amblyopia and amaurosis have been observed with cortical tumors, unaccompanied by choked disk. These are all to be attributed to a lesion of the visual centre; and, when located in the frontal lobe, the lesion must be regarded as indirect. The amaurosis or hemiopia, with tumors of the centrum ovale (39 cases, or 31 per cent.), probably always implies a transmitted lesion of the cortical visual centre. Of the two cases of hemiopia, referred to in Table X., one is used by Exner and Nothnagel as documentary evidence in support of the theory of a visual centre in the cortex of the occipital lobe, but it is placed by Bernhardt among the tumors of the lobes. The total percentage of blindness is higher with tumors of the cerebellum than with those of any other locality, except the corpora quadrigemina. Out of ninety-one cases there are forty-one with some de-

[1] See Seguin's papers on Hemiopia in Journal Mental and Nervous Disease.

gree of blindness (45 per cent.). Of these, twenty-three, or nearly half, are without choked disk; the blindness being therefore due to the direct action of the tumor upon some visual centre. It seems most probable that the centre then affected is that of the corpora quadrigemina; the influence being transmitted through the superior cerebellar peduncles. The high percentage of blindness in the two localities so especially liable would be shown, therefore, to have the same significance. Tumors of the pons and medulla also determine amaurosis otherwise than by choked disk, through direct upward pressure upon the corpora quadrigemina. The direction of the transmission is the same as for the upper (unassociated) nucleus of the motor-oculi nerve, which lies just below the corpora quadrigemina. Out of a total of fifty-one cases for medulla and pons together, there are fourteen cases of amblyopia or amaurosis, or twenty-seven per cent.

TABLE X.—Lesions of Vision (in 369 Cases).

Seat.	With Choked Disk.					Without Choked Disk.					Negative	
	Total No. of cases.	Hemiopia.	Amblyopia.	Amaurosis.	Per cent.	Hemiopia.	Amblyopia.	Amaurosis.	Per cent.	Total per cent.	Choked disk.	No choked disk.
Central gyri	...	...	...	1	...	...	...	...	...	...	3	3
Parietal	...	...	...	1	...	...	3	2	...	...	...	1
Frontal	...	...	...	...	...	...	1	...	...	...	...	1
Occipital	...	...	...	...	...	...	...	...	...	...	...	1
Temporal	...	...	...	1	...	...	...	...	...	...	1	...
Entire cortex	56	...	...	3	5.0	...	4	2	10.5	16.0	4	4
Centrum ovale	124	...	4	11	12.0	2 {1 occipital. 1 frontal.}	9	13	19.0	31.5	12	7
Basal ganglia	26	...	...	...	...	...	4	1	19.0	19.0	2	1
Cerebral peduncle	10	...	1	...	...	...	2	...	...	...	...	...
Pons	30	...	1	3	13.0	...	6	...	20.0	33.0	2	1
Medulla	21	...	...	...	...	...	1	3	...	19.0	2	1
Cerebellum	91	...	4	14	19.5	...	9	14	25.0	45.0	11	3
Corpora quadrigemina	11	...	2	3	45.0	...	3	1	36.0	81.0	1	...
Total	369	...	12	37	...	...	42	36	...	...	38	19

Total lesions of vision = 118 in 362 cases = 31 per cent.

To judge from this table we should infer that the chances of amaurosis in brain tumor were exactly equal, whether choked disk existed or not; but that the chances of amblyopia were three times as great without the choked disk as with it. This probably means that if choked disk occur, the impairment of vision which may have been initiated independently of it, by the direct influence of the tumor, will rapidly increase to complete blindness; whereas, without this local complication, the visual defect may for a much longer time, or even altogether, remain partial and incomplete.

Hearing, Taste, and Smell. — All these special senses together are less frequently affected than is vision alone. Out of a total of three hundred and sixty-nine cases of brain tumor, lesions of vision existed in one hundred and eighteen, or thirty-two per cent. But in a total of five hundred and fifty-four cases (which include Ladame's), hearing, taste, and smell are altogether only affected in sixty-seven, or twelve per cent. In forty-six out of these sixty-seven cases the patient suffered from either tinnitus or deafness, the latter rarely complete. In twenty-six out of the forty-six, thus in more than half, fifty-six per cent., the tumor was situated in the cerebellum. This fact tends to confirm, if need be, the recent anatomical demonstration, which traces the central fibres of the acoustic nerve to the cerebellum. By far the highest percentage of disturbance of hearing is exhibited by tumors of the corpora quadrigemina. It is singular that reports of tumors of the frontal lobes so rarely mention symptoms indicating lesion of the olfactory tracts. It would seem that an indirect influence or diffused pressure is insufficient to pervert the sense of

smell; that is only affected by actual disorganization of the tracts. In a few cases, anosmia, associated with frontal headache, psychic disturbance, and absence of motor or sensory paralysis, has been a valuable symptom which correctly pointed to tumor in the frontal lobes. But anosmia has also been observed with a tumor of the supramarginal convolution. The sense of taste, though controlled by two medullary nerves, usually escapes injury, even with tumors of the medulla.

TABLE XI.

LESIONS OF SPECIAL SENSES (561 CASES—369 FOR VISION).

Seat of tumor.	Hearing.	Per cent.	Taste and smell.	Per cent.	Vision.	Per cent.
Cortex (59 cases)	1	1.5	4	7.0	9	16.0
Cerebrum ovale (192 cases)	8	4.0	3	1.0	39	20.5
Basal ganglia (41 cases)	2	4.0	...	...	5	12.0
Peduncle (3 cases)	...	...	...	...	3	...
Corp. quad. (13 cases)	4	30.0	...	...	9	70.0
Cerebellum (167 cases)	26	15.0	2	1.0	41	25.0
Pons (56 cases)	3	5.0	9	16.0	10	18.0
Medulla (30 cases)	2	6.0	3	10.0	4	14.0
Total	46	8.0	21	3.5	120	21.0

Disturbances of Language. — These symptoms, formerly confounded either with symptoms of mental alienation, or else with difficult articulation caused by tongue paralysis, have, during the last two decades, acquired an extreme interest and importance. The discovery that a patient may retain other mental conceptions, yet lose that of spoken or written speech; furthermore, that the generic defect may be again resolved into several modes, namely, aphasia proper, agraphia, alexia, and simple "word-blindness"; this discovery has immensely widened the horizon of ideas in regard to the physiology and pathology of the

brain, and has enriched the symptomatology of all brain diseases, including tumors.

According to the most recent classifications, cases of aphasia must be divided into two classes : sensory aphasia, or aphasia of reception ; and motor aphasia, or aphasia of transmission. In the first class, the patient fails to understand the significance of language, which he hears merely as an unintelligible sound. In the second, if uncomplicated, he understands entirely what is said, but is unable to express himself in words. The lesion of sensory aphasia is located in the posterior part of the first temporal convolution of the left hemisphere ; the lesion of motor aphasia is in the locality described by Broca, the third left frontal convolution, or the insula of the same side. Agraphia, or inability to write, which may complicate alexia (inability to speak), or exist as an isolated symptom, has been referred to lesion of the second frontal convolution. In the absence of such lesion, the aphasic patient who cannot use spoken language, will remain able to express himself by writing.

The various forms of aphasic symptoms help to localize the seat of a tumor, as of other focal brain lesions ; but they do not of themselves serve to distinguish between tumor and softening ; and, therefore, can only point to tumor when associated with other symptoms. Unlike other symptoms, aphasia does not require discussion in relation to the relative liability of different regions of the brain ; for, as above shown, the presence of one or the other form, at once tends to assign the lesion to a definite locality.

From the foregoing analysis of the causation and especial probabilities of diffuse and focal symptoms, it is possible, in a given case, to answer the two ques-

tions: first, Is there a brain tumor present? second, In what part of the brain is it situated?

I. Existence of Brain Tumor.—Although a tumor of the brain may develop either during childhood or adolescence, let us suppose it to have begun its growth in an individual of middle age, who perhaps has shown a tendency to tuberculosis. In such a case we can assume that the clinical picture will be somewhat like the following: For weeks, or months, the patient will suffer from persistent or periodic headache, usually localized at one spot; the pain is peculiarly severe, and is increased by percussion. After a time there will be attacks of vomiting, which sometimes coincide with the most intense paroxysms of pain, and sometimes do not. These attacks, furthermore, seem to bear no relation to the character of the food taken, or to the condition of the digestive organs; they do seem, however, to be dependent upon changes in the position of the body, as, for example, from the recumbent to the upright position. As in the case of sea-sickness, the attacks are sometimes incoercible. They are associated with vertigo; and, in turn, the vertigo may occur independently of either the headache or the vomiting. It is apt to occur at intervals, and is often chronic in character. After the symptoms which have just been enumerated have lasted for a variable length of time, the patient's gait becomes uncertain; he reels or staggers, or shows a tendency to fall forward or backward. This tendency sometimes increases until complete loss of equilibration renders the patient unable to stand, though he may be entirely free from paralysis. The muscles of one side of the face or of one arm begin to twitch, or even to be agitated by clonic spasms, which

may either persist all the time, except during sleep, or else may recur in periodic paroxysms, followed by paresis, gradually increasing to paralysis in the same muscles or in others, *e. g.*, in the arm or leg, after twitching of the muscles of the face. The progress of the paralysis is apt to be interrupted by one or more convulsions, or by attacks of apoplexy or of loss of consciousness; or one of these may usher in the first signs of paralysis, which, at the outset, may be complete, facial, monoplegic, or hemiplegic. Paræsthesia or anæsthesia is next likely to manifest itself in the paralyzed limbs, or on the side of the face opposite to these. Afterward the symptoms succeed one another in about the following order: alternate paralysis of cranial nerves and extremities; deviations of the eyeballs, isolated or conjugate; dilatation of the pupils, ptosis, much more rarely appearance of Bell's paralysis; occurrence at this time of diplopia, hemiopia, or amblyopia, gradually increasing to complete amaurosis; much more rarely deafness or anosmia, and the discovery of choked disk before or after the development of ocular symptoms; progressively increasing modification of psychic character—at first marked irritability, then impairment of mental powers, loss of memory, apathy or hallucinations, maniacal excitement, and melancholic insanity; before or at the same time with the appearance of this mental change, there will be lesions of speech, dysarthria, aphasia, or word-blindness, the two latter often suddenly developed, as after an embolus, the first proportioned to the degree of tongue paralysis, and gradual. A patient presenting the foregoing assemblage of symptoms, all progressively increasing, has, with very great probability, a brain tumor. In

addition is to be noted the freedom from pyrexia, and usually from changes in the rhythm of either pulse or respiration. The gradual, sometimes rapid, emaciation, the fact that acute accidents, though often followed by an exacerbation of existing symptoms, or even by the first appearance of new ones, have nearly always been preceded by others which have established themselves insidiously, are circumstances important to the diagnosis.

This being the general picture of the disease, individual cases are framed by the special emphasis of one or more symptoms, or the obliteration of others. The individual peculiarities depend upon (1) the locality of the tumor, (2) upon its rate of growth, (3) upon its complications, (4) and, only to a very slight extent, upon its nature.

Peculiarities Due to Locality.—These may be divined approximately from such an analysis as has already been given of the symptoms proper to lesions of each given locality. The *a priori* judgment must, however, be modified in view of the tendency of tumors to encroach, in growing, upon territories adjoining their original seat, and also in view of the frequent diffusion of their influence beyond any situation which they may occupy.

The following summary of symptom groups is arranged in the order of characteristicness. It does not correspond to the order of frequency of locality, which, as indicated by the combined tables of Ladame and Bernhardt, would be as follows:

Centrum ovale	192 = 29	per cent.
Cerebellum	162 = 27	"
Cortex	74 = 11	"
Pons	56 = 8	"

Basal ganglia	36	5	per cent
Medulla	30	4	"
Corpora quadrigemina	13	2	"
Cerebral peduncle	10	1	"
Extra cerebral (including pituitary gland)	71	11	"
	649		

1. *Tumor of Cerebellum Involving Middle Lobe.*—Vertigo, vomiting, and headache, early, severe, and prolonged; latter often occipital; epileptiform convulsions of great violence, but not often repeated; choked disk early, preceding amaurosis, but also followed by this; deafness; ataxic loss of equilibrium, tendency to fall forward or backward; absence of motor or sensory paralysis; intelligence clear till toward the end, when apathy gradually deepens to coma.

2. *Tumor of Lateral Lobe of Cerebellum Pressing on Pons.*—Similar symptoms to 1, but complicated late in the disease by hemiplegia or hemianæsthesia, or both, or by alternate paralysis. Distinguished from pontine tumors by marked ataxia preceding paralysis.

3. *Tumor of Pons, Lower Half.*—Uncertainty of gait, rather than ataxia, succeeded by isolated paralysis of third, or sixth, or seventh, or twelfth nerve, not preceded by symptoms of irritation in the muscle which it supplies; or else alternate paralysis, passing into incomplete paraplegia or general paralysis; permanent conjugate deviation of the eyes; amaurosis in a third, choked disk in a fifth, of the cases; entire absence of convulsions; headache, vomiting, and vertigo milder than in cerebellar tumor, or absent, but intelligence affected in half the cases.

4. *Tumor of Upper Part of Pons.*—Combination

of symptoms proper to cerebellum and pons, as lobe of cerebellum is frequently compressed. Isolated rather than conjugate paralysis of the third nerve; paralysis of the facial on the same side as hemiplegia; irritation of the trigeminus, sometimes of motor root, occasioning trismus; or of sensitive root, causing neuralgia on the side opposite to the hemiplegia. Sudden death is especially frequent in tumors of the pons.

5. *Tumor of Cerebral Peduncle, or of Interpeduncular Space.*—Diffuse symptoms mild or absent; complete hemiplegia, including buccal branches of facial, usually accompanied by hemianæsthesia on same side; paralysis of motor-oculi nerve, causing divergent strabismus on side opposite to hemiplegia, this frequently passing over to opposite side.

6. *Tumor of Cerebral Cortex, or Upper Part of Centrum Ovale.*—Epileptiform convulsions, frequently repeated, but often brief and of moderate severity. Headache usually frontal, possibly on one side, severe at first, apt to gradually lessen; choked disk infrequent, vomiting and vertigo much less marked than with tumors of posterior fossa. Spasmodic twitchings or clonic convulsions, in face or limb muscles, followed by dissociate or monoplegic paresis increasing to paralysis, rarely accompanied by anæsthesia. Paralysis of tongue, hemiopia, or peculiar visual hallucinations, sometimes seen when tumor is in occipital lobe, but also, (once at least) when in frontal. Aphasia, especially in connection with right hemiplegia. Word-blindness sometimes without paralysis, the tumor then being in the temporal lobe. Psychic disturbance in about half the cases (forty-nine per cent.).

7. *Basal Ganglia, or Lower Part of Centrum Ovale.*

—Complete hemiplegia, often followed by rigidity, thus resembling the hemiplegia of hemorrhage, except in regard to the more gradual development of the former. Sometimes associated with complete permanent hemianæsthesia. Often complicated, late in the disease, with symptoms of intraventricular effusion; thus, for example, there are convulsions, retraction of the head, loss of consciousness, slow pulse, contracted pupils, as in acute hydrocephalus.

8. *Corpora Quadrigemina.*—Individual cases can with difficulty be distinguished from tumors of the cerebellum. In the calculation of the probabilities, however, the much greater frequency of cerebellar tumors (twenty-seven per cent. of the whole number as compared with two per cent.) is not to be forgotten. With tumors of the corpora quadrigemina, however, the percentage of headache, though high, is less so than with those of the cerebellum; the percentage of vomiting is higher; convulsions seem to be so rare that their presence in a doubtful case would turn the scale against the corpora. The proportion of cases of choked disk is high, and of amaurosis, as also of psychical defect, higher than for tumors of any other locality (eighty-one per cent. of cases are amaurotic; seventy-seven per cent. present psychic symptoms). Divergent strabismus from paralysis of the motor-oculi is sometimes present, and is then very characteristic.

9. *Medulla.*—All symptoms ill-defined; they resemble those of tumors of the pons. Dysarthria, dysphagia, and irritation of the cardiac and respiratory centres are more frequent. Paralysis of the vocal cords has been observed in a single instance.

Besides the localities already mentioned, the clini-

cian must always inquire whether the tumor whose existence is suspected does not spring from the cranial bones or the dura mater lining them. Tumors of the anterior, middle, and posterior cranial fossæ excite symptoms which approximately resemble those belonging to the cerebral organs reposing in the same spaces.

Anterior Fossa.—This comprises two regions, the sella turcica, with the pituitary gland and the optic chiasma, and the part anterior to this, upon which repose the frontal lobes of the hemispheres. In the latter position tumors may cause exophthalmia on one or both sides. Apart from this, the characteristics are similar to those of tumors of the frontal lobes. Hemiplegia, spasms, and epileptiform attacks are in the background, while headache and vomiting play about the same part that they do in other tumors. It is asserted that the most notable symptom is psychical in character, viz., a peculiar childish alteration of character.

Sella Turcica.—Although tumors in this region involve the pituitary gland, it is impossible to assign any special symptoms to lesions of that organ, whose functions are so entirely unknown. Patients suffer from severe frontal headache, but they manifest a striking apathy and drowsiness, without marked motor or sensory paralysis, or any disturbance of speech. From generalized pressure on the chiasma results double progressive amblyopia or amaurosis.

When the tumor bears other relations to the chiasma, it produces some species of hemiopia. If anterior to the chiasma, it produces a double temporal hemiopia, from compression of the fibres coming from the inner half of each retina. On one side of the

chiasma the tumor would only affect the outer fibres of one retina, or, if situated further back, so as to compress an entire optic tract after decussation—that is, all the fibres from the homonymous parts of the two retinæ, as, for example, the right or the left halves of both eyes—it would produce homonymous hemiopia.

Middle Fossa.—The most characteristic symptoms of a tumor of the middle fossa depend upon lesion of the trigeminal nerve. Thus, there may be unilateral anæsthesia of the face, unilateral weakness of the masticatory muscles, and, finally, if the Gasserian ganglion be injured, neuro-paralytic keratitis of one eye. Through the medium of the chorda tympani branch of the trigeminal, the sense of taste may also be paralyzed.

In addition to these most characteristic paralyses, there is frequently paralysis of the motor-oculi nerve, of the facial (five times out of nine), and of the acoustic nerve—the latter usually by penetration of the tumor into the internal auditory canal of the petrous bone. With the facial paralysis there is degenerative electrical reaction. With these marked positive symptoms are associated certain negative symptoms—absence of motor or sensory paralysis in the extremities, absence of convulsions, absence or mildness of the headache or the vomiting.

Posterior Fossa.—Tumors of this region cannot with any certainty be distinguished from those of the medulla, pons, or lower segment of the cerebellum. In one case a peculiar conjugated deviation of the eyes has been observed, the right eye turning upward and outward, the left inward and downward. This deviation recalls Magendie's experiment of section of the right lateral peduncle of the cerebellum; and it is probable that this organ was involved in the tumor.

Amaurosis or amblyopia exists in one third of the cases—that is to say, even more frequently than in tumors of the cerebellum. But, in any individual case, this symptom can serve no purpose of diagnosis.

Parts of Brain in which Tumors are most Frequently Latent.—Complete latency implies absence of all symptoms; incomplete latency implies absence of focal symptoms only. The localities in which the latter condition is characteristically observed are also those in which tumors may most often be completely latent. These localities are: The temporal, occipital, or even, but less easily, the frontal lobes of the cerebral hemispheres, provided the central gyri are not indirectly affected; the parts of the centrum ovale corresponding to these regions, and hence untraversed by fibres from the pyramidal tract; the lateral lobes of the cerebellum, the thalamus opticus, and the lenticular nucleus. Finally, it is possible that in any portion of the brain a tumor may remain latent, providing it grow slowly enough.

Differential Diagnosis.—The epileptiform convulsions dependent upon cerebral tumor differ little or not at all from those of functional epilepsy. They are, however, often slighter, or at least the loss of consciousness is much less profound. The headache, on the contrary, is chiefly noticeable for its extreme intensity and persistence, in which respect it exceeds even nervous headaches. The vomiting is also noticeable for its violence, and for the absence of any other symptoms of disordered digestion, such as furred tongue, epigastric uneasiness, etc. The diagnosis in regard to these symptoms ultimately depends on their combination, and on their association with paralysis or with psychical symptoms. Conversely,

the psychical symptoms of tumor are distinguished from pure mental alienation chiefly by the existence of these physical signs; also by their greater vagueness, which renders precise psychiatric classification difficult or impossible.

It is by no means always easy to decide whether a patient with cerebral symptoms is suffering from a diffused or focal disease, and in the diagnosis of tumor it is necessary to exclude meningo-encephalitis, progressive general paralysis, chronic basal meningitis, hydrocephalus, cerebro-spinal form of multiple sclerosis, and locomotor ataxy.

Tubercular meningo-encephalitis, which easily lasts three months, has, it must be remembered, a duration not inferior to that of many tumors, and many of the symptoms are identical: violent headache, convulsions, vomiting, neuritis optica, changes of character, monoplegic paralysis, and spasms. In the diffused inflammation, however, these paralyses are transient and variable, a condition sometimes, but rarely, seen in tumor. The disease, moreover, is always attended by more or less fever, by more marked variations in the pupils, by a slow, hard pulse, by obstinate constipation, by retraction of the abdomen, aud by vasomotor symptoms. When a tubercular tumor is associated with diffused inflammation, it is masked by the symptoms characteristic of the latter.

A tumor of the medulla may especially simulate progressive general paralysis by producing a diffused paresis without distinct paralysis, embarrassment of speech, depression of mental power, headache, and unequal dilatation of the pupils. A tumor, however, is indicated by the occurrence of amaurosis, convulsions, vomiting, localized paralyses; while the diffused

disease is characterized by the appearance of ambitious delirium, and by the peculiar trembling of the lips. Tumors of the sella turcica may be closely simulated by chronic basal meningitis, which is most frequently situated in exactly the same locality, and also involves the same nerves. It is distinguished by the occurrence of descending optic neuritis, unattended by symptoms of intracranial pressure. In young children, premature closure of the fontanelles, with blindness, would point to meningitis ; enlargement of the head, to tumor.

Hydrocephalus may also have choked disk, and is usually associated with depressed mental capacity. Slow enlargement of the head belongs either to this disease or to tumor, in young children. The rolling down of the eyes and subsequent retraction of the head point to an effusion. Ventricular effusions are not infrequent complications of tumor, especially of tubercular tumor ; but the idiopathic disease does not occur except in very young children.

Multiple cerebro-spinal sclerosis may for a time simulate tumor, the disease being characterized by headache, vertigo, disturbances of speech and of vision (diplopia and amblyopia), and by the occurrence of apoplectiform attacks, followed by incomplete hemiplegia. The latter, however, are rare in tumor, but are apt to be frequently repeated in sclerosis. In sclerosis, on the other hand, there is an absence of convulsions and of motor paralyses, except after apoplectiform attacks. Instead, there is a diffused loss of power, with muscular rigidity, absence of vomiting, and choked disk. Finally, the appearance of the characteristic tremor of the limbs is a positive symptom which decides the question in favor of sclerosis.

Locomotor Ataxy.—It may occasionally be difficult to distinguish the ataxia of cerebellar tumor from that of tabes spinalis. But in the tumor the patient has a staggering or reeling gait, like that of a drunken man, and there is no sign of ataxy in either upper or lower extremities when the patient is in a horizontal position (Althaus). The alterations of sensibility, characteristic of tabes, are absent in tumor, and most of the positive symptoms of tumor are absent in tabes.

Abscess of the brain, which, from its focal symptomatology, is ranked by Ball and Krishaber with tumors, sometimes simulates typhoid fever with pronounced cerebral symptoms. The absence of either pulmonary or abdominal symptoms, however, may prevent error, until the appearance of some localized paralysis decides the diagnosis. Two focal diseases of the brain often resemble tumor extremely—cerebral hemorrhage and softening.

Cerebral Hemorrhage.—The onset of the paralysis is sudden, instead of being slow and insidious, and the paralysis is usually at once complete. But the cranial nerves are rarely affected, with the exception of the facial; vomiting, headache, vertigo, and choked disk are absent, as are also mental symptoms after recovery from the apoplectiform shock. Hemorrhage into the meninges, which scarcely ever occurs except in children and old people, does not resemble tumor in any of its symptoms with the exception of convulsions.

Softening.—The diagnosis from tumor is often extremely difficult when the softening is, from the beginning, chronic in character. Lesions of special senses are much less frequent in softening, and choked disk is rare; so also are lesions of cranial nerves, vomiting and convulsions; while the headache is less

circumscribed and intense. Contractures of paralyzed limbs are more frequent. Psychic alterations are marked, but are of a different character from those of tumor. There is emotional instability instead of irritability, dementia rather than the depression and apathy of tumor.

A diagnosis of the nature of the tumor can rarely be made.

Carcinoma is often indicated by the rapid progress of the symptoms, and by signs of multiple foci successively developing. Perforating tumors are almost invariably malignant—carcinoma, sarcoma, or osteosarcoma. The tumor is nearly always primary, and destroys life before it has occasioned cachexia.

Tubercular tumor often complicates tubercular meningitis, or is complicated by it. In either case the focal symptoms are much obscured by those of the diffuse disease. When isolated, a tubercular tumor may be suspected from the youth or scrofulous constitution of the patient.

Gummata. — Their diagnosis principally depends upon the presence of other signs of syphilis. The evolution is relatively rapid, and the invasion of drowsiness and coma may be hastened by the coexistence of diffused endarteritis.

Glioma.—This remains the most probable when the diathetic tumors have been excluded. It not infrequently develops after a blow on the head, and then seems to result from chronic inflammation of the neuroglia.

Intracranial aneurisms occasion symptoms which are indistinguishable from those of neoplasms proper. It is the basilar artery which is most often affected, and the symptoms then resemble those of tumors of the

pons. But all the arteries are liable to be the seat of this lesion. It is said that headache is more diffuse and more intense than with any other tumors, while vomiting is less frequent. Sudden attacks of loss of consciousness often occur, due undoubtedly to inequalities in the distension of the tumor, and consequent variations in the brain pressure.

Aneurisms of the posterior communicating artery occasion symptoms of motor-oculi paralysis (ptosis, external strabismus, fixed dilatation of the pupil), and, finally,—the effect spreading to the corpora quadrigemina,—amblyopia. When the aneurism is seated on the internal carotid, the sensitive root of the trigeminus may be affected ; hence neuralgias or anæsthesia. Aneurisms of the carotid which communicate with the cavernous sinus are characterized by exophthalmia, and a susurrus which is heard when the stethoscope is applied over the eyeball (case—Gruening).

The termination of aneurismal tumors is peculiar, being always by rupture and sudden death, with the symptoms of intracranial hemorrhage.

Abscesses of the brain comport themselves like an acute tumor. Their evolution is habitually much more rapid, and their progression much more regular than that of neoplasms. There is sometimes fever, but often this is absent, and the tumor may be entirely latent for some time. An abscess is always to be suspected when localized cerebral symptoms develop in the course of an otitis media. Extremely chronic cases of this aural affection sometimes pass into an acute exacerbation, during which the cerebral membranes become infected through the roof of the tympanum, through the fenestræ, or through the auditory canal.

PROGNOSIS.—The prognosis of cerebral tumor is not modified by the diagnosis of either the seat or the nature of the tumor, unless the latter can be shown to be syphilitic. Gummatous tumors sometimes yield with remarkable rapidity to the mixed treatment for syphilis. All others are invariably fatal, but after a longer or shorter lapse of time, and with somewhat different modes of termination. Thus, as has been said, aneurisms terminate by rupture, and death occurs with all the symptoms of cerebral hemorrhage. In the majority of cases the patients die in coma, gradually developed from a condition of apathy and drowsiness. These states are associated with continually increasing brain-pressure, which often results in œdema. Sudden death is not uncommon, and is dependent upon inhibition of the cardiac centre. This sudden death may occur as an accident after the most variable duration of the disease; but even the mode of death which seems to indicate the natural evolution of the morbid process, leaves a most variable time for this to be accomplished. The patient sometimes dies as early as ten or even eight weeks from the appearance of the first symptoms; in other cases, these have been prolonged for ten years.

PATHOLOGICAL ANATOMY.—The histological structure of many cerebral neoplasms, including aneurisms, does not differ from that of the same growths in any part of the body. Tubercular tumors, like miliary tubercle, always start from the lymphatic sheaths of the blood-vessels, beginning in a local accumulation of adenoid elements. Gliomata are a species of sarcomatous tumors, which are peculiar to the brain. They were described as neuromata, until Virchow demonstrated that they contained no nerve elements,

but developed from the neuroglia. The glioma may consist almost exclusively of cells, and is then called a medullary glioma; or it may contain a large amount of connective tissue, which either remains soft and of the myxoma type (myxoglioma), or becomes hard, fibrous, or even cartilaginous (fibrous glioma). Finally, some among these tumors are so rich in vessels as to have acquired the name telangiectasic gliomas. All develop from the neuroglia. The tumor appears as a grayish mass, becoming pink or red as vessels develop in it. If these are numerous, apoplexies may take place into the substance of the tumor. The three forms of malignant tumor of the brain are sarcomas, carcinomas, and melanoid tumors. The first are closely related to the gliomas, arising like the medullary variety of the latter; the chief difference consisting in the greater size of the cells and the larger amount of intercellular substance. Cancer of the brain is nearly always encephaloid, primary, and not infrequently congenital. The growth is rapid, and the size ultimately attained by the tumor is in inverse proportion to the vital importance of the part of the brain in which it is seated. Enucleation of the tumor is impossible. Cancer of the upper part of the cerebral hemispheres not infrequently perforates the dura mater, and even the skull. Conversely, cancer of the eyeball, usually melanotic, constantly tends to penetrate the brain.

Melanoid tumors are forms of carcinoma in which the tissue is infiltrated with pigment. Their most frequent seat is the eyeball, the pigment being derived from that of the choroid.

Hydatid cysts are found in the brain, but they usually remain latent, especially if small and multiple.

Other forms of cyst are not infrequently formed by hemorrhagic effusion, by softening of brain-tissue from extensive necrobiosis, or by the softening of myxomatous tumors.

Complicating Lesions.—With glioma, congestion and hemorrhage in the vicinity of the tumor are the most frequent complications, the latter often being the cause of death. The tissue around the tumor is often the seat of an inflammatory softening. Effusion into the ventricles is often caused by compression of vessels which return blood from the choroid plexus. Such effusion is common with tubercle, and then may depend on granular thickening of the ependyma.

A zone of non-inflammatory softening surrounds most tumors. It depends upon necrobiosis of nerve-tissue, from localized obstruction to the circulation, and œdema. When this softening is extensive, functional regions quite different from those actually occupied by the tumor become involved. This circumstance, as has often been shown, by complicating the symptoms, often materially obscures the diagnosis.

When the fibres of the pyramidal tract have been affected by the tumor, descending degeneration of secondary sclerosis may set in, and even reach the lateral columns of the cord. This is, however, much less common than after hemorrhage; and, correlatively, late rigidity is correspondingly rare. Conversely, the appearance of rigidity in limbs paralyzed from the effects of a cerebral tumor, often indicates that hemorrhage has been excited in its vicinity.

Treatment. — There is no radical medical treatment except for gummata, and for these the mixed treatment sometimes yields brilliant results.

The suggestion has recently been made to remove tumors situated near the surface of the brain by a surgical operation. The suggestion has been carried out in the following remarkable case (Bennett and Godlee, London *Lancet*, December 20, 1884):

Farmer, aged twenty-five. Symptoms lasted three years, beginning one year after a blow on the head. Paroxysmal twitching of the left side of the face and tongue. General convulsions, then local spasm of the left arm, cessation of the convulsions, paresis of the arm, twitching of the left leg, violent headaches, attacks of vomiting, double optic neuritis. Patient trepanned at point of skull corresponding to upper (?) part of the fissure of Rolando. Dura divided, ascending frontal convolution exposed, and found to be distended. Incision of one fourth inch disclosed hard glioma, of the size of a walnut. Patient at once relieved of lancinating pains, vomiting, and convulsions, but paresis of left leg increased. Improvement up to twenty-first day. Then rigor, fever, nausea, and pain in head; hernia cerebri. Death on twenty-eighth day. On post-mortem examination, signs of meningitis at lower portion of the wound, spreading downward toward the base of the brain on the same side, the whole of which was inflamed and covered with plastic lymph.

III.

NOTE ON THE SPECIAL LIABILITY TO LOSS OF NOUNS IN APHASIA.[1]

SOME months ago, it occurred to me that it would be interesting to ascertain in how many cases of aphasia the defect bore upon any particular part of speech or mode of speaking. For this purpose I examined the records of one hundred and sixteen cases, and found that, among them, in seventeen, the patient had only lost the memory of noun substantives, or the faculty to employ these in voluntary speech. They were replaced by a periphrase, in language often quite fluent. Among the ninety-nine remaining cases, in only two was any other part of speech systematically affected. In one, the patient had lost the adjective, but she had also lost the noun. In the other, the patient had lost control over pronouns, some of which, however, were used, but improperly, and only employed the infinitive of verbs. The seventeen cases are as follows:

CASE I.—Broadbent describes a patient, aged 77 at the time of death, who was seen at intervals between 1878 and 1883. His infirmity dated from a slight and fugitive attack of right hemiplegia, predominating in the face, and accompanied by hemianæsthesia. There was at first a somewhat general disturbance of speech, which finally became restricted to the loss of nouns. This defect persisted five years. During all this time the patient never uttered a noun but once or twice, and then inappropriately; could say any thing else, and employ long phrases, so that they

[1] Read at the Neurological Society, 1886.

did not contain a noun. When he wished for any thing he would say, "Please give me the one."[1]

CASE II. was another of Broadbent's, similar in all respects to the first, except that the patient could not read.[2]

CASE III. was also a patient of Broadbent's, a gas inspector, aged 59. The first complaint of this patient was, that he found himself unable to read, and could not remember names of places, persons, or things. Pointed to legs and arms, and said that he forgot the names of these. On another occasion, said that he could not recollect the name of this, taking hold of his coat. The doctor said "trousers." He said at first "yes," but then said "coat." Asking him afterward to name his finger, he muttered "coat, hat, boot," then was silent. I suggested thumb. He said, "yes, thumb," but afterwards "finger." This same patient was able to give a lucid description of an accident which had happened to him seven or eight years before.[3]

CASE IV. is less striking, because the entire faculty of speech was much more compromised: The patient had a few favorite routine expressions, as "Ca va bien; un petit mieux." He could not repeat the name of the objects shown to him, and made fruitless efforts to do so. If he were told the name, however, he would recognize it as correct; make a sign of affirmation, and observe, "oui, c'est ça." But he could not repeat the word himself.[4]

CASE V.—(Case of Dr. Allin, reported by Drs. Ball and Seguin.[5]) The patient, after a third attack of cerebral accidents, recovered power of speech to a considerable extent, but had much difficulty with proper names and common names. Of a glass of milk he would say, "That is something to drink." Would have flashes of fluency on various subjects. With the progress of convalescence, the patient's vocabulary increased.

CASE VI.—Trousseau describes an eminent lawyer who had the habit of frequently forgetting the name of the thing about which he wished to speak. Addressing his wife, he would say,

[1] Med. Chir. Trans., 1872. [3] Ibid.
[2] Med. Times and Gazette, 1885. [4] M. F. Balzer, Gaz. Méd. de Paris, 1884.
[5] Archives of Medicine, 1881, vol. iv.

"Give me then my—my—*sacré matin*, my—you know very well." Then he would raise his hand to his head. "You want your hat?" "Yes, my hat." On another occasion, as he was going out, he rang the bell. "Give me my um—*sacré matin!*" "Your umbrella?" "Yes; my umbrella."[1]

Case VII.—Bateman,[2] quotes from Bergman (*Zeitschrift für Psych.*, 1849) the case of a man, who, after a fall, lost the memory of proper names and common substantives. He retained memory of verbs, and was able by means of periphrases to express his meaning.

Case VIII.—The same author also quotes from Graves (*Dublin Quarterly*, Feb., 1851) the case of a farmer, who, after an attack of hemiplegia, could no longer employ nouns in his speech, though he always remembered the initial letter.

Case IX.—This was one observed by Bateman himself, three years after accidents, which consisted exclusively in the sudden loss of speech. At the time of observation, the patient was able to talk, but not to use substantives except incidentally. Thus on being shown a purse, remarked: "I can't say the word; I know what it is; it is to put money in." Here it is noticeable that, although the noun which was required as the object of the proposition could not be remembered or pronounced, yet another noun, money, referred to incidentally, could be named.

Case X.—Lasegue[3] describes a priest from Canada, aged 65, who could relate his own history fluently, but used no nouns, or only with the greatest difficulty.

Case XI.—Lordat relates the case of the naturalist Broussonnet, who only retained the use of two nouns, soir (evening), which indicated the future; and juments (mares), by which he referred to a lady and her daughter. He replaced all other nouns, common or proper, by periphrases, or by a series of adjectives. Thus he called one friend, "He whom I love well"; and another, "The great, good, modest one."[4]

Case XII.—At an Academy discussion in 1873, Bouillaud mentioned a man, known to Cuvier, who had lost the memory of

[1] Peter, Gaz. Hebd., 1864. [3] Annales Méd. Psychol., 1877 (Soc., Feb. 26th).
[2] Aphasia. [4] Quoted by Bernard, "De l'aphasie," 1885, p. 185.

nouns, but was able, nevertheless, to compose phrases regularly and completely.[1]

CASE XIII.—Piorry quotes the case of an abbé who had lost the memory of nouns. He would say "give me my—that which one puts on the—" then point to his head, showing that he meant his hat, or else "give me that which is worn to clothe one's self."

CASE XIV.—Bernard quotes another case from Bateman, where the patient, instead of scissors, would say, "that with which one cuts," and for window, "that by which one sees," or "that where it makes light." In this second expression, as in another case already quoted, the patient used a noun incidentally (light), but could not do so with deliberate intention.

CASE XV.—A patient of Gairdner's called Monday, "the first working day," his aunt, "his nearest relative on the mother's side."[2]

CASE XVI.—This is described by Dingley. Five weeks after a slight attack of hemiplegia, patient was obliged to use circumlocutory phrases to describe objects. Thus, whenever shown the picture of a camel, he said, "Egypt long way."

CASE XVII.—Lichtheim relates a case of word deafness, where the patient talked a good deal in a flowing manner, though with some tendency to repetition of the same phrases, but he always had the greatest difficulty in naming objects, and assisted self by descriptive phrases. Thus, for wine he would say, "that is strong;" for water, "that is weak."[3]

From the foregoing list are excluded the much more numerous cases on record where the patient used the wrong nouns to express his meaning. For obvious reasons are also excluded cases where the entire vocabulary was extremely restricted.

To any one who first begins to examine the records of published cases, it might seem as if a much larger

[1] Compt. rend. Acad. des Sciences, t. lxxvii., 1873.
[2] Arch. de Méd., 1866, 6e S., t. viii.
[3] Brain, January, 1885.

number could be collected of any given peculiarity. But all remarkable cases have done service many times, by being quoted over and over again by different authors, so that much care in verification is required in order to avoid repeating one case as several.

The peculiar form of aphasia under consideration has attracted much attention. Lasègue declared that the loss of the noun, "the substance of the discourse," was the most characteristic circumstance of aphasia.[1] Bouillaud called attention to this peculiarity in his communication to the Academy in 1873; Chevreul, following, offered an explanation of the fact. Falret, in 1866,[2] Bateman, in 1870 (quoting also an explanation by Osborne), Voisin in the "Nouveau Dictionnaire"; Bernard, in his monograph in 1884, all note that if any grammatical part of speech is systematically lacking to aphasics, it will invariably be the noun. Kussmaul,[3] I believe, makes a separate category of such partial aphasias, as do also Broadbent[4] and Lichtheim.[5] "The loss of the noun," observed Ross, "is the most marked form of sensory aphasia."[6] "It is evident that the amnesia progresses from the special to the general. It first affects the individual, the proper names, then the names of things which are the most concrete, then all substantives used in an adjective sense,—finally, adjectives and verbs which express qualities, states of existence, and actions." "The idea of quality is the most persistent, because it is the first acquired and forms the basis of our most com-

[1] Loc. cit.
[2] "Dictionn. Ency." Art. Aphasie, 1866.
[3] "Die Störungen der Sprache in Greisen."
[4] Medical Times and Gazette, June, 1884, also Med. Chir. Trans., 1872.
[5] Loc. cit.
[6] "Handbook Dis. Nerv. Syst.," Philadelphia, 1885.

plicated conceptions" (Ribot, "Das Gedächtniss"; translated into German from the French).

The existence of this feature of language defect has sometimes seemed to conflict inexplicably with the common belief that children in learning to talk, learn nouns first. It is then supposed that the noun must be that part of speech which becomes the most firmly "organized" in the brain, and should therefore be the last to disappear when the brain is injured. Yet the reverse is certainly observed.

The partial, or, as we may call it, the noun defect, is observed in amnesia (sensory aphasia). Case VI., from Trousseau's clinics, illustrates amnesia without aphemia; the patient forgot the names of objects, but when told this name, he recognized it as correct, and was able to pronounce it.

Case XIII. is precisely similar. In other cases, it is not stated whether the patients were able to repeat the name which they were unable to remember. The impression is conveyed in a majority of the histories that this could not be done. When the spoken word was nevertheless understood, it is to be inferred that there was no serious defect on the motor side of the speech mechanism, aud that the receptive, sensory side, was only incompletely injured. For in focal lesion of the auditory centre, spoken language sounds like gibberish to the patient. And where the power exists to repeat the word under the influence of the immediate stimulus from the auditory centre, this implies that the path between that and the co-ordinating centre of articulation is intact, and also that the latter centre is not seriously damaged.

Two general inferences must be drawn. 1st. That the lesion in these cases of partial defect is relatively

slight. 2d. That it involves the paths connecting the auditory with the concept centre, or those which associate the latter with the motor co-ordinating centre. These conditions would be fulfilled by a moderate diffused lesion or perturbation of the conducting tracts B M or A B in Lichtheim's schema.

"The lesion is amnesia," observes Lichtheim, "is not focal, but appears in more diffused morbid processes, or where cerebral circulation is deficient." The records of autopsies are not as useful as might at first be supposed, in solving the problems of this partial amnesia. To some of the most interesting recorded cases, no records of autopsies are appended. In the others, the lesions found belonged either to a period of disease from which the patient had partially recovered when he exhibited the partial defect, or to an exacerbation which preceded death, aggravated the symptoms, and determined the fatal issue.

Thus it is really more profitable at present to examine the question from the point of view of the mechanism of the naming process, considered in both its psychological and physiological aspect. Around the naming process have ranged some of the most celebrated controversies of philosophy. Whether the names of things, *i. e.*, nouns, were used first, as Dugald Stewart[1] asserts; or whether the first words were verbs, and indicated action, the theory of Adam Smith; whether common names were evolved from proper names, or the reverse; whether a class name represented a real existence apart from the individuals composing it; or whether it stood for a real concept,

[1] According to Dugald Stewart, the primitive men on seeing a wolf coming would cry, "wolf, wolf." According to Adam Smith, they would shriek, "he comes," and point to the beast in explanation (quoted by Max Müller, "Science of Language," p. 31).

a conceivable notion of the mind ; or whether it were strictly a sign for a collection of attributes, these being alone conceivable,—such questions as these racked the brain of humanity centuries before the cerebral localization of speech was dreamed of. That the existence of a class name proved the existence of a real abstract being, an archetype upon which the individual members of the class were modelled, was the doctrine of the realists of antiquity and of the middle ages.

But no one any longer supposes that the words man, or horse, or table corresponds to abstract but real beings, and this famous doctrine has no bearing upon the psychology of the naming process. It is otherwise with the second or conceptualist doctrine. This is constantly to be found cropping out, often unconsciously, from the most positivist descriptions of the mechanism of speech. In these, English physiologists, at least, usually assume the necessity of explaining, first, how a concrete or general idea or concept is formed from sense impressions, then how a name becomes attached to this idea. The mode of attachment is sometimes very oddly expressed. Thus, Ferrier is quoted by Hammond as saying : "The ideas of which words are the articulate symbols have no relation to that part of the brain where words are remembered, except by associating fibres." [1] We may justly ask what is meant by attaching an idea to any part of the brain. We might as well talk of connecting the time occupied by the run of a railroad train with the space it goes over. Broadbent,[2] in an analysis of the mechanism of speech, in many respects most

[1] West Riding Reports, 1874 (quoted by Hammond, "Dis. Nervous System," Eighth Ed., 1886, art. "Aphasia").

[2] "Med. Chir. Trans.," 1872, vol. iv.

admirable, observes: "The conception or idea of external objects is gradually formed by the fusion of the visual, tactual, and other impressions to which it gives rise. This idea is associated with an auditory impression which has been used to designate it." If for the term of "conception" we should substitute the other, "mental image," little would be lacking in Broadbent's description, at least from the standpoint of our present knowledge. Yet danger lurks in the term "mental image" also. The younger disciples of the purely materialistic school sometimes commit themselves to unintelligible absurdities by attempting too much precision in the history of "mental images."

Thus Mlle. Skwortzkoff, author of a good thesis on aphasia, and of an article on word-blindness, describes the evolution of the spoken word as follows: "Every object strikes several senses at once, and causes the development of as many sensitive images, whose totality constitutes the idea we have made for ourselves of this object. The impression forms in a first centre into a sensation, and this in a second cortical centre forms an image. The different sensitive images are *transmitted* towards the centre for the formation of words (foot of the third frontal convolution and surrounding parts), where the totality of these images takes its formula, its name. This name, by means of fibres of transmission, reaches the medulla, whence the nervous fibres animating the diverse parts of the apparatus of phonation project it outwards.[1] But what is a name that it can be thus transmitted on nerve fibres like a messenger on the string of a boy's kite?

In this connection it is well to remember the caution of Hughlings Jackson: "A method which is founded

[1] Mlle. Skwortzkoff, Archives de Neurologie, 1881, t. II.

on classifications which are partly anatomical and physiological, and partly psychological, confuses the real issues." [1] These mixed classifications lead to the use of such expressions as that an idea of a word produces an articulatory movement; whereas a psychical state, an "idea of a word" (or simply a "word") cannot *produce* an articulatory movement, a physical state . . . We must keep these several sides of our subject apart, considering now the psychical side—speech,—and at other times the anatomical basis of speech.

Speaking, then, exclusively on this anatomical basis, we may say, with Broadbent, that impressions made by the object upon the various perceptive centres of the brain, fuse together, after converging upon some cell area intermediate to these centres, into a complex impression of this object. When the object has been named at the time it was perceived, an auditory impression is made simultaneously with the visual and tactual impressions, and this fuses together with the rest. Now it is possible to revive the mental image of the object by reviving any one of the original impressions, or even the memory of these. Among these means of revival, that of the auditory impression or name is so frequently made, and has so many conveniences, that it becomes the habitual sign of the rest; and the name is used to draw into the consciousness of the person speaking or of the person addressed all the secondary or revived impressions of the sense attributes of the object.[2] "The word," observes

[1] Brain, Oct., 1878. "On Affections of Speech," Hughlings Jackson.

[2] "Whatever performs the office (of directing our attention to particular elements in the perception) is virtually a sign; but it need not be a word: the process certainly takes place to a limited extent in the inferior animals; and even with human beings who have but a small vocabulary, many processes of thought take place habitually by other symbols than words. . . . In many of the fam-

Whitney, "is simply the survival of the fittest, among a variety of resources, (gestures, etc.) for effecting the same purpose, namely, the mental revival of the attributes of an object.[1] Thus, as Taine remarks, the association of a name with an object creates a *couple*, formed on the one hand by an auditory sign, on the other by the group of attributes with which the sign is associated. Of this couple, either member has the power of bringing the other into consciousness; and, the first extension of mental processes becomes possible when the sign may be substituted for the thing, and handled apart, like a mathematical symbol.[2]

In these descriptions, the word "impression" is used with an intentional vagueness, to cover the unknown molecular processes which take place in the cortical sensory centres, in the intermediate cell areas, of, as Broadbent suggests, the non-sensory, the superadded convolutions, and in the innumerable tracts of nerve fibres which associate these together. Of these processes, we can only frame to ourselves a schematic representation. While for some purposes the term "images" answers well enough in this schema,[3] for others it is misleading, and the conception of a molecular vibration answers much better. It certainly is

iliar processes of thought, and especially in uncultivated minds, a visual image serves instead of a word." John Stuart Mill. "Examination of Sir William Hamilton," 1865, vol. ii., p. 73.

[1] Whitney, "Life and Growth of Languages," 1882. The author remarks that speech has the preference over gesture, even when it is less forcible and explicit, because it leaves the hands free.

[2] On Intelligence. Am. Transl., 1872, p. 6. "In the formation of couples, such that the first term of each suggests the second term; and in the aptitude of this first term to stand wholly or partially in place of the second, so as to acquire, either a definite set of its properties, or all those properties combined, we have, I think, the first germ of the higher operations which make up man's intelligence."

[3] It is constantly used by Meynert.

much more in accord with such analogies for nerve action as we are almost compelled to draw from the physical phenomena, sound, light, and electricity.

The phenomena of musical combinations afford a guide at least for the schematic description of the name-evolution. The sound of the spoken name is certainly produced by air vibrations, which mediately impress the auditory nerve, and conceivably throw its molecules also into vibration. We may represent to ourselves these vibrations as continued to the cortical auditory centre, and there determining others, which, according to the special lines of intercellular fibres that are traversed, cause what Broadbent has called the specialized grouping of cells. These are not, of course, displaced in the nerve mass, but brought diversely into relation with each other, in the same way as battery cells scattered through a laboratory may be diversely grouped according to the wires included, at any given moment, in the circuit. As far as our present data carry us, such a specialized vibration in the auditory centre would suffice to bring the sound of the spoken word into consciousness. The "fusion" of this vibration with others analogous, coming from the visual and tactual centres is, as we must conceive it, analogous to the fusions of small groups of musical vibrations into larger groups, producing more complex sounds. This complex vibration, occurring in the so-called concept centre of Lichtheim, the superadded convolutions of Broadbent, does not "*produce an idea*"; it is itself the physical side or substratum of one phenomenon of which the conscious impression, idea, image, or concept, is the psychic aspect. The concept again, is not, as Sir William Hamilton declared, something conceivable by the understanding,

though not by the imagination[1]; 'but so far as it means any thing, it is the *equivalent* of the mental image, or the psychic aspect of the complex vibration. This mental image differs from each sensory image by the very fact of its complexity, and also by its probable formation in non-sensory portions of the brain. It is these anatomical localities, and not the ideas, which are connected with the sensory centres by association fibres. Finally, the auditory impression or vibration does not become a name in the auditory centre; but only after it has become an integral part of the complex, fused vibration, whose psychic aspect is the idea of mental image. Hence a name in an unknown language is gibberish. The same consideration shows that the name is not affixed to the idea of an object after that has been separately elaborated. It is possible, it is true, to perceive an object whose name is unknown to the percipient. But, if the latter wish to communicate any impressions of this object to another person, he must make use of some sign to indicate it, and the sign, though but an indicative gesture, is already the essence of the word, and is simply replaceable by a verbal sign when that shall have been suggested. In the absence of communication, actual or potential, there is no language.

Although a concrete name be the sign for a real mental image, composed of the remembered attributes of the object named, a general name is not. It is here that the modern philosophic doctrine of nominalism becomes identified with the modern physiological doctrines of speech and thought. The philosopher may declare that there is no abstract conception in the mind, the physiologist that there is no material

[1] "Lectures on Metaphysics."

image in the brain, no matter how refined and etherealized. It is impossible to have an abstract conception of a triangle that shall be free from any peculiarities of some individual triangle, as a scalene or isosceles, etc. But it *is* possible to abstract the property of three-angledness from a class of figures, of which each individual possesses this, though possessing other properties besides. It is this property or attribute that is recalled to the mind, and which the mind is capable of contemplating apart by means of the special verbal sign—triangledness—attached to it. "Thus," observes Hamilton, "a sign is necessary to give stability to our intellectual progress, to establish each step in our advance as a new starting-point for our advance to another beyond. A country may be overrun by an armed host, but it is only conquered by the establishment of fortresses. Words are the fortresses of thought." [1]

The internal mental image becomes realized in speech through further propagation of these (supposed) cerebral vibrations toward the point where they can determine such grouping of nerve cells as can secondarily regroup cells in the ganglionic centre immediately presiding over organs of phonation,—that is, towards the corpus striatum. All recent testimony tends to localize this point of convergence at the foot of the third left frontal convolution. The considerations which precede, suffice to show, however, the absurdity of regarding this convolution as the "seat of the faculty of language." Broca himself only claimed that lesion of this convolution was followed by "loss of the memory of the means of co-ordination that are employed to articulate words." [2]

[1] Quoted by Mill, loc. cit., p. 68.

[2] P. Broca, Bull. Soc. Anatom., 1883, t. viii. (quoted by Bernard, loc. cit., p. 175).

The far greater extension given to-day to the total cerebral mechanisms employed in speech, render superfluous the criticisms upon Broca's doctrine which are based on the discovery of lesions of parts of the brain other than this convolution, and which have been found to co-exist with some form of aphasia.[1]

I have not found any record of cases which show a loss of power to articulate names, when it was clear that these could be spontaneously recalled by the patient, and when, at the same time, other parts of speech could be articulated.

When an object or a class of attributes constituting an abstract conception can be recalled to mind, but its name cannot, it is evident that the visual and tactual perceptions of the objects have persisted, while the auditory impression, or else its point of fusion with the rest, has been effaced. Chevreul says that this has happened because less attention has been paid to the name than to the sense attributes of the object. Ross, following Hughlings Jackson, says that names disappear first in the dissolution of speech, thus in the mildest cases, because they are less well organized knowledge than that of simple relations.[2] I think there is another reason which may be rendered clear by considering the primitive development of speech. It is highly improbable that this began in the use of either nouns or verbs, but rather in conglomerates, shorter or longer, which constituted an entire proposition. Children, in learning to speak, use words at first with precisely this complex significance, and it is a matter of accident whether the word employed be a noun,

[1] Thus Hammond, in the latest edition of his treatise, reproduces a table published by Seguin in 1868 (Quart. Journ. Psychol. Med., Jan., 1886), containing eighteen autopsies called in favor of Broca's theory, and thirty-four against. This merely refers to the cases with and without lesion of the third frontal convolution.

[2] Loc. cit.

verb, adjective, or even a preposition. I knew a little boy, extremely intelligent, but who, at the age of two years, could only say five words, yet contrived to express himself wonderfully well by gestures. But one of his few verbal signs was "hard-a-lee," an expression that he had learned while sailing, and which he habitually used either to refer to a sail-boat, to urge a wish to go sailing, to announce his possession of a boat to a new-comer, etc. The verbal conglomerate was not learned first because it was simple or easy, for it was neither; but it belonged to the circumstance that had made the most forcible impression on the baby's mind.

According to Renan, many primitive languages abound in conglomerate expressions. The Greenlander treats an entire phrase like a single word, and conjugates this word like a simple verb. Among the majority of the North American Indians, continues the same author, the composition and agglutination of words is pushed to an almost incredible extent. Each phrase of these languages is only a verb, in which all the other parts of the discourse are inserted.[1] In the successive experience of both the individual and the race in the acquisition of speech, the order would seem to have been as follows: 1st. There are the sensory impressions made by the qualities of the object. 2d. A proposition arises in some one's mind to be communicated about this object to another person by means of a verbal sign, more or less extensive in significance, but probably always at first unique. 3d. There is a gradual breaking up of this conglomerate sign into words occupying special relations to each other.

[1] "De l' origine du Langage," Sixth Edition, 1874, p. 156.

Whitney observes that the establishment of a clear distinction between the noun and the verb especially marks the genius of the Indo-European languages, and it is not nearly so well marked in others.[1] In the development of these languages, words originally betoken qualities—the most general circumstances,—which are gradually specialized and individualized towards concrete objects. Thus, although the hypothesis be provisionally useful for the purpose of analysis, it is probably not really correct to say that the process of naming ever consists in fusing a verbal sign merely with the sensory impressions of a single object. The conglomerate verbal sign was evolved from original interjectional sounds, under the pressure of a strong desire of communication with a fellow-being. For this very reason, the sign must always have implied a proposition concerning the object referred to. So long as the primitive man simply recognized the wolf, and took his own precautions for defence, there was no language. Language began when men began to concert together for defence against a common enemy. The very least that could then be said was, "There is the wolf," or "the wolf comes," complete propositions involving a subject and a predicate, but both probably expressed together by a single conglomerate sign. This sign represented the fusion of an auditory impression, not only with the group of visual impressions which made up the general mental image of the wolf, but with the visual impressions of events in which the wolf took part. At the present day, though the original conglomerate be broken up into separate words, the phrase still retains its unity in thought. If from lesion

[1] "Life and Growth of Language."

of the associating fibres through which diverse impressions may be fused, this unity is weakened, and the phrase threatened with dissolution, the part which first tends to disappear is that which is most easily replaceable by the visual image. This is certainly the part of the phrase of conglomerate sign which indicates the object itself. The speaker can point to it when in sight, can describe it by periphrases when it is out of sight; but such replacement is possible for nothing else in the proposition. As long, therefore, as speech is possible at all, it will express by verbal signs those parts of the proposition which cannot be expressed in any other way, while the name which can be diversely suggested is forgotten as a simple sign. As Kussmaul observes: "Conceptions of persons and things, are more loosely associated with their names than the abstractions of their conditions, relations, and properties. We can represent persons and things to ourselves without their names; . . . but more abstract ideas are only grasped by the help of words. So adjectives, pronouns, adverbs, prepositions, conjunctions are much more closely associated with thought than are substantives. We may suppose that in the reticulum of the cortex far more numerous excitation processes and combinations are necessary to shape an abstract than a concrete idea." (Die Störungen der Sprache.)

Ribot adds: "The persistence of verbal signs in memory is proportioned to their degree of organization—that is, the number of repeated and registered experiences."

Temporary forgetfulness of a name is, as is well known, not at all uncommon among quite healthy people. Any one, by observing himself closely in

these cases, may recognize that the difficulty of recalling the name seems to be directly proportioned to the clearness of the visual image of the object. As an example: I found myself the other day telling a person to go down on the piazza, and stammering over the word "piazza," while I was at the same time picturing to myself the locality with unusual distinctness.

The patients who recall the names of objects that are incidentally imbedded in the phrase describing an object whose name they cannot recall, illustrate the theory here advanced. When such a one says, upon seeing a purse, "I know what it is, but cannot name it; it is to put money in," the noun, "money," is merely part of an adjective phrase which might be expressed "it is money-containing."

The name recalls the properties of money so faintly that the visual image of this object cannot triumph over the verbal sign and obliterate it. But the object in view, the object of the entire proposition, excites a visual impression so much more powerful than the auditory sign belonging to it in the verbal conglomerate—the phrase,—that this sign is obliterated. It is not, of course, that the visual impressions or memories are absolutely increased in strength; they become relatively stronger simply because the mechanism for the revival or for the association of all verbal impressions is damaged, and these, therefore, are weaker.

It seems to me that this theory is much better grounded than that which attempts to distinguish between the words which "are better organized in the brain" and those which are less so. No auditory sounds, however highly specialized, are words, until they are understood as the signs of things, or of the relations of things. And no words are, in themselves,

any fixed part of speech, but only exist as words in the relation they occupy to the mental grouping of the moment.

It is this relation which first disappears in sensory aphasia, while enough of the mechanism for recording verbal auditory impressions remains to enable the patient to recognize a name pronounced before him. The association of this verbal sign with the visual impressions of an object may be so much damaged that revival of the one in consciousness will not recall the other. The psychological difficulty depends on physical injury to the anatomical tracts which connect the visual and auditory centres.

In the conglomerate mental image framed of the object and of a proposition concerning it, there will persist the reminiscence of the sense impressions of the object and the auditory signs used for enunciating the proposition. These signs have never been connected with any particular visual impression, but only with a series of relations whose memory is registered or organized in the concept, supra-sensory centre. In the milder forms of sensory aphasia the paths between these intellectual centres and the auditory centre on the one hand, and the motor centre on the other, are presumably intact; no dislocation takes place between the auditory signs and the series of relations to which they correspond. The name of the object is, however, entirely dislocated from its habitual associations; the impulse or vibration which passes from the visual centre goes directly to the concept centre, without fusion with any impulse coming from the auditory centre. The final mental conglomerate of the proposition, therefore, which is to be expressed, consists partly of reminiscences of sense im-

pressions, partly of revived verbal signs, instead of being composed entirely of verbal signs, as is normal. The verbal signs which remain in the conglomerate are repeated by the articulatory mechanisms which receive the appropriate stimulus to functional cell grouping. The visual reminiscences of the object cannot be expressed by these mechanisms, any more than waves of sound could be reproduced by the retina, or waves of light by the auditory nerve. This substantative portion of the conglomerate proposition can only be expressed by gestures or by visual signs. Such signs must have served the purpose of expression before any auditory signs had become specialized into speech. They serve such purpose again when auditory signs have become disassociated with objects on account of lesion of the anatomical paths through which visual and auditory impressions may fuse together. The dissolution of speech follows the reverse order of its development; the concrete names, the last framed, fail first.

It has been suggested to me by a friend who listened to the exposition of the foregoing theory, that, in accordance with it, abstract nouns, as "love," "patriotism," "virtue," should be retained by the aphasics in question, because they are associated with no definite visual image, but with series of relations. It would be interesting to test this suggestion.

IV.

CASE OF NOCTURNAL ROTARY SPASM.[1]

(Reprinted from *The Journal of Nervous and Mental Disease*, July, 1880.)

THE case of rotary spasm I have asked permission to describe to the Society, exists in a boy of three years of age, remarkably chubby, and presenting the appearance of the most perfect health. Since his birth, he has never had any illness except a mild attack of scarlatina, which occurred six months after the first development of the present affection. This began at the age of eighteen months—thus eighteen months ago. The mother then noticed that, after the child had been asleep for a couple of hours, he would turn over on his right side, drawing the right arm above his head, and applying the left hand over the left ear. Once in this position, he would begin to oscillate his head on the pillow from right to left, in a perfectly rhythmical manner. The oscillation would be maintained for about half an hour, and then the child slept quietly again. From the time this phenomenon was first observed, no night passed without its occurrence; but for the first six months, the rotary movements were not very rapid—did not last very long—and thus did not attract any great attention. They were ascribed to a morbid habit of no especial

[1] Read before the New York Neurological Society.

significance. During the last year, however,—thus ever since the attack of scarlatina,—the oscillation has increased in rapidity, in duration, and even in extent. At first exclusively confined to the head, the rotation has successively involved the shoulders and the trunk. At first confined to half an hour, it now habitually lasts several hours, and even the whole night.

It is noticed that if after the paroxysm had begun at nine and lasted an hour, the child was awakened, he would sleep quietly until midnight, but that then the movement would recommence and become most violent between five and six in the morning. After that he would fall into a very heavy sleep, and instead of awakening early, as usual with children of this age, the boy would sleep till 7½ or 8.

Change of locality would generally diminish the violence of the nocturnal movements for a few nights. But they would then regain their original intensity; often the thumping of the crib as the child rolled from side to side would make a noise sufficient to keep awake the mother or nurse in an adjoining room.

In the morning following a night thus agitated, the child would seem to be in nowise fatigued, and certainly retained no recollection of his nocturnal gyrations. He never could be induced to repeat them voluntarily, though he had become impressed with the solicitude they excited, and would often threaten to "shake his head," in order to tease his mother. He would even, when requested, lie down in the position in which the paroxysm habitually occurred, on the right side, with the right arm above the head, and the left hand applied to the left ear. But in this position, while awake, no attack occurred; although the invariableness with which the attack during sleep was pre-

ceded by the assumption of this position, suggested some connection between it and the rotary spasm.

In view of such a possible connection, on the first occasion on which I witnessed the phenomenon, I turned the child on the left side. The movements immediately ceased, and on that occasion—a nap taken in the daytime—did not return. The mother reported that this manœuvre had often been tried, and always with the effect of temporarily checking the rotation. The child resisted the turning with considerable force, and, as soon as left to himself, turned over on his right side, and recommenced his oscillations.

It was noticed that the paroxysms rarely occurred when the child slept in the daytime, or if they did they were of very moderate severity. But this fact seemed dependent on the other, that the rotations only took place during a very sound sleep, and after this had lasted about two hours.

On the first occasion on which I saw the child, however, he had been brought from some little distance in the country, was very tired, and readily went to sleep at noon. The rotations of the head began in half an hour.

Starting from the attitude of repose on the right side, the head was thrown to the left and a little upwards, with a slight jerk, so that the face looked upwards and to the left, the occiput downwards and to the right. It was then immediately restored to its former position, so that the face worked downwards and to the right, the occiput upwards and to the left. These positions were alternated seventy-two times in a minute, and were rhythmically regular. The movement from right to left, which seemed the initial movement, was always jerking, the movement of restitution, from left to right, was not. Accompany-

ing the oscillations of the head were twitchings of the eyelids, and apparently oscillations of the eyeballs.

The first half of the oscillation was necessarily effected by sudden, brief contraction of the right sterno-cleido-mastoid, together with the clavicular portion of the right trapezius, and probably also of the splenius. (Duchenne, pp. 2, 714, 715.) The second half of the oscillation, or the movement of restitution, necessitated similar contractions on the part of the homologous muscles on the left side. Faradization (after Duchenne's method) of the right sterno-cleido-mastoid muscle of a healthy woman lying on the back, rotated the head upwards and to the left with a jerking motion, in a manner entirely resembling the first half of the oscillation in our case.

During this oscillation the forehead of the child was slightly contracted, and a very slight shade of distress seemed to be impressed on the child's features. The pulse was 87, soft and regular. The temperature of the left parietal region, alone accessible with the patient in position, was 94.5° (94.4° G.). After watching the oscillation for fifteen minutes, and observing no change, I turned the child carefully on the left side. All movements immediately ceased, both of head and eyelids, and the child continued to sleep tranquilly. The mother attributed this result, unusual in her experience, to the unusual degree of fatigue caused by the journey.

Immediately on turning the child on the left side, I noticed a considerable change in the pulse. It increased in fulness and strength, and in frequency to 115. In five minutes it had fallen to 99, and became much softer. In ten minutes it had returned to the original rate of 87.

While the child lay on the left side the parietal temperature of the right side was measured, and found to be 93.° (93.59° G.). The temperature of the occipital region was 95.° (91.94° right, G.). Thus this portion of the head was 3.06° higher than the average temperature for the occiput as given by Dr. Gray.

After about fifteen minutes the child was turned over again on the right side; but the oscillation did not return. No change was noticed in the pulse, such as had been observed after turning the child in the opposite direction: it remained soft, and at 87 beats in a minute.

A few weeks later I had an opportunity of observing a nocturnal paroxysm. This began punctually at 9, the child having fallen asleep at 7. At first the rotation was confined to the head, and resembled that already described. But a little later, after some interruption, the movement changed. With the left hand over the left ear, the child began rotating the entire upper half of his body, softly, rhythmically, about seventy times a minute. The head moved with the shoulders and trunk; the lower limbs remained quiescent. A little later in the evening this rotation was accompanied by a crooning cry, also rhythmical.

The child had the air of rocking himself to sleep to his own lullaby.

This cry was a feature in the case that had only recently been added. It reminded me of one that I once heard uttered by a child during the clonic period of an eclamptic convulsion. The child was suffering from intermittent fever, and very often had convulsions at the time of the chill. Whenever these convulsions were severe, the automatic inarticulate crying would begin, and gradually shape itself into a tune,

which was always the same, namely, "Pop goes the Weasel." The inarticulate crooning of the child whose case we are now describing was modulated into no definite melody. But, like the above, it seemed to depend upon a succession of clonic contractions of the constrictors of the glottis, analogous to the contractions affecting the other muscles.

During this nocturnal attack the face of the child became very much flushed, as had not been the case during the first two hours of sleep. The mother reported that this flushing was a constant accompaniment of the rotation, though it had not existed during the mild attack I witnessed in the daytime.

The temperature at the occiput was 96¾°—thus still higher than had been observed on the previous occasion.

In the daytime, careful examination of the child, especially in regard to motor incoherences or ataxia, or to any disturbance of the special senses, yielded completely negative results. The expression and gestures were vivacious and intelligent. The articulation, however, was more defective than usual for children of three years old. Until the age of two and a half, its speech was said to have been completely unintelligible.

The head of the child presented no marked abnormality of shape. The forehead, however, was projecting, and the palate much arched.

The inquiries in regard to the faculty of equilibration and to the sense of hearing, were especially suggested by the resemblance which the rotatory movements of the child bore to those which, in animals, follow unilateral section of a lateral peduncle of the cerebellum, or of the horizontal branch of the semicir-

cular canals. Such mutilation is apt to be followed, not only by rotations of the head, but also by rotations of the entire trunk, and are accompanied by oscillations of the eyeballs. Clinically speaking, there can be no doubt that the morbid condition belongs to the group of choreiform affections, of which the salaam convulsion or spasmus nutans, and the saltatory convulsion, are the types.

In the spasmus nutans, both sterno-cleido and mastoid muscles are affected, and hence results a nodding movement of the head. "But when," observes Eulenburg, "there is unilateral clonic convulsion of the same muscles, the movements are rotatory. The point of the chin is turned towards the sound side; the occiput is drawn down; the ear and mastoid process approached to the clavicle of the affected side."

As already noticed, this movement can be exactly imitated on a healthy person who is lying down, when one sterno-cleido mastoid is intermittently faradized. Eulenburg further notices that these clonic spasms are often not isolated, but are accompanied by contractions of the muscles innervated by the facial, trigeminus, and oculo-motor nerves. The movements are sometimes very slow, sometimes as rapid as 100 a minute.

Erb describes a rotation of the chin from one side to the other, occasioned by alternate spasm of both sterno-cleido mastoids. He asserts that the bowing movement caused by an exactly synchronous action of the same muscles, is much less frequently observed.

Soltmann, in Gerhardt's new encyclopædia, also describes two forms of "spinal-accessory convulsion." It consists, he says, in a double or rhythmically alternating contraction of the antagonists, whereby the

head is now turned from one side to the other, or else the chin is alternately depressed or elevated.

In 1850, Dr. Willshire read a paper on The Eclampsia Nutans, or Salaam Convulsion, in which he stated that there were only four well authenticated and detailed cases on record, those namely described by Dr. Newnham in 1839. Of these cases, three children died and one recovered. The latter was sixteen months old when she began to have attacks of "head nodding" three times a day. The paroxysms rapidly increased in number and severity, and the convulsive movement extended to the trunk, which was forcibly bowed, sometimes as often as 140 times a minute. The paroxysm seemed to occasion considerable suffering, and was followed by exhaustion and drowsiness. After three months, the child lost the ability to crawl she had previously acquired. A month later, the attacks began to come on during sleep, from which the child would awaken with a violent scream, and in a spasm of the whole body, the head being first thrown back and then bowed violently to the feet, which were also drawn upwards. Six months from the beginning of the attacks, the child fell into a comatose sleep which lasted some hours, and from this date improvement commenced. The clonic convulsions ceased altogether, but the intellectual development of the child was arrested, so that at three years she was no more advanced than at two.

In Dr. Newnham's other cases the children became hemi- or paraplegic, and completely idiotic.

In Dr. Willshire's case the child was only six months old when the bowing movements of the head began. These were repeated fifty times a minute, and were so extensive that the head was made to touch the

knees. These paroxysms always occurred after sleep, and were severe in proportion to the intensity of the sleep. They never occurred during sleep. Occasionally they were replaced by general epileptiform convulsions. This case recovered under a treatment of purgatives, blisters behind the ears, iodide of potassium and quinine.

Dr. Bidwell's case, reported in 1852, terminated as unfavorably as did the three cases of Dr. Newnham. The nodding movements began at the age of six months, occurring three to four times a day, being at first repeated only a few times in the course of a minute or two. At the age of a year, the frequency and intensity of the paroxysms had increased, consisting of thirty or forty convulsive movements in rapid succession. By this time it became evident that the mental development was very much retarded, if not wholly arrested. The slight nod of the original paroxysm increased to the true Oriental "salaam," in which the head was suddenly drawn quite down to the floor, often bruising the forehead and lips. Later, epileptic convulsions occurred, and at the end of second year the child was hopelessly idiotic and epileptic. She died at the age of twenty-six months. No autopsy recorded.

In 1850 Dr. Faber reports a case, in a child of three years, whose health had begun to suffer only three months before coming under observation. The nodding paroxysms came on suddenly, after much complaint of headache and drowsiness, and were accompanied by strabismus. The nodding paroxysms merged into epilepsy, and the child became idiotic.

In a second case observed by Faber, the patient, a child of six years, was severely frightened by falling

down a well. After that he seemed to droop; his sleep was restless, and he frequently cried out in it. One day, having been scolded by his father, he began to nod his head violently, while at the same time the face was distorted. The nodding movements occurred about eighty times a minute; the paroxysm lasted three or four minutes and returned several times a day. At its close the child was evidently much fatigued. A condition of stupidity supervened, analogous to that occurring in chorea. Ultimately, however, the child improved under the administration of iron.

In 1867 Dr. Morgan published in the *Lancet* a case of *rotatory cramp* of the head, observed in a man thirty-eight years old. Since childhood he had suffered from headache and from a choreatic affection of the right arm, which prevented him from writing. A rotatory cramp of the head developed after exposure to cold. While in bed, or while sitting or standing, this was very slight, but so soon as patient began to walk, the chin was convulsively drawn to the shoulder, the head inclined to the opposite side, while severe pain was felt both in the neck and also in the occiput. The occipital tuberosity was painful on pressure. Dr. Morgan considered that the convulsive rotation was principally effected by the left sterno-cleido-mastoid and right trapezius muscle; and, acting on this theory, he cut the left spinal-accessory nerve, paralyzing the trapezius and sterno-mastoid on the left side. After the operation, although the trapezius, with the splenius and complexus, still remained affected, the patient was able to walk without a convulsion, if he took the precaution to hold the clavicular fibres of the trapezius between his fingers.

In 1868 Henoch described cases of spasmus nutans,

not limited to the head, but involving the entire upper part of the body. In one case the nodding convulsion alternated with lateral movements of the head from right to left. Nystagmus often coëxisted. One of Henoch's cases was ameliorated in fourteen days. The other terminated, suddenly, in death.

The latest recorded cases that I have been able to find are by Kropff, reported in 1877, in an inaugural dissertation. The first was in a consumptive woman, attacked by the convulsion during the puerperal state. The convulsion was accompanied by pains in the region of the left occipital and the frontal nerves, and consisted in nodding of the head seventy or eighty times a minute. At the same time the head was turned a little to one side. Voluntary movements were possible, and the clonic contractions could be passively overcome. The morbid condition was cured by tonic treatment and by iron.

The second case was in a man, in whom the head was thrown first directly backwards, then forwards. The spasm lasted only a second, but was repeated twenty or thirty times a minute. It was succeeded by a tonic spasm of the constrictors of the glottis, so that the breathing was arrested for a few seconds; then by clonic spasm of the pectoral and deltoid muscles, causing involuntary movements of one arm. From these cases it is evident that the spasmus nutans and the spasmodic affections allied to it may be either purely functional disorders, or else symptomatic of organic cerebral disease, as in the earlier cases described by Newnham, Willshire, and Bidwell. As far as can be at present ascertained, the case I have described belongs to the first category. But it differs from them, and from all of which I can find a record, in two

important particulars. 1st. The rotatory paroxysms occur in the recumbent position and during sleep; while in the other cases recumbency has quieted, and sleep arrested, the paroxysms. 2d. The rotatory spasm in our case exists only so long as the body is maintained in a certain position.

Is it possible to draw any diagnostic inference from these two facts?

The occurence of the paroxysm during profound sleep, its intensity during the early morning, and the profound sleep by which it is followed, the flushing of the face, and the inarticulate cry accompanying the rotatory spasm, suggests many analogies with epilepsy, which, in the absence of organic lesion, might justify us in classing the affection as an epileptiform rather than as a choreiform neurosis. For the occurrence or non-occurrence of morbid symptoms during sleep is well known to be one of the most striking points of contrast between the symptomatology of epilepsy and of chorea.

It may be mentioned in this connection that the only medicine which has in any way seemed to control the paroxysm is bromide of potassium; this even when given in small doses. On last seeing the child, he was ordered a mixture of bromide and chloral: ten grains of bromide and five of chloral twice a day—twenty grains of bromide and five of chloral at night. For ten days after taking this medicine the child slept without the rotation. Then a plentiful crop of acne developed, and the mother interrupted the medicine. The paroxysms returned. The medicine was repeated, but this time its effect was much less marked. The paroxysms were not interrupted, but they were diminished in intensity—did

not begin until twelve or one o'clock—on many nights were omitted. This diminution persisted even after the medicine was interrupted.

The second peculiarity in our case which distinguished it from the general type of the spasmus nutans was the peculiar position of the body, which seemed to be a necessary condition for the occurrence of the spasm. The forcible turning over to the right side, the curving of the body, with the convexity to the left, the bending downwards of the head, offered a close resemblance to the forced attitude assumed by animals after lesions of certain parts of the brain. Section of one lateral peduncle of the cerebellum, or unilateral section of the medulla, carried down as far as the level of the tuberculum acusticum, will each be followed by an assumption of this peculiar attitude.

The convex curving of the body may be directed towards the wounded or the sound side.

It is said to have been first observed by Magendie after the unilateral section of the medulla. But, as is well known, Magendie, and many other observers after him, from Serres and Flourens to Ferrier, have been able to produce, by section of a lateral peduncle of the cerebellum, rotatory movements of the head and trunk, which succeeded to this fixed attitude, and were directed from the healthy to the wounded side. The rotary movements observed in our case resemble these in every respect, even in the fact that they are directed from the side towards which the body is concave, towards the side at which it is convex, thus the presumably morbid side.

Such rotary movements are known also to follow section of the horizontal branch of the semicircular

canals on one side, as I have myself had an opportunity to observe in pigeons. But were lesions in these two localities alone capable of producing forced rhythmical oscillations, the data from experiment would fail to explain all the circumstances of our case. For in it the movements began with a simple lateral jerking of the head, such as may be produced by faradic irritation of the sterno-cleido-mastoid muscle, and seemed therefore to depend exclusively on irritation of the spinal-accessory nerve. At this stage of the affection it constituted a typical "clonic accessorius convulsion." And the proximate seat of the irritation must have been in the nucleus of the 11th nerve, situated in the medulla. Now Magendie, who has first described the rotary movements and the fixed position determined by unilateral section of the medulla, has established that the latter lesion will also determine movements of rotation of the head and trunk around the longitudinal axis of the body. Recently Curschmann has asserted, that section of the peduncle of the cerebellum never determines the movements, but only the forced attitude, if the section be made at a certain distance from the medulla, and infers that the peduncular lesion is only effective in virtue of a secondary influence upon the medulla. With this view Eckhard entirely agrees.

However this may be, there is no question that unilateral lesion of the medulla will be followed by this peculiar phenomenon, and it is unnecessary, in the absence of personal experience, to adduce more authorities in proof of this.

Clinically, the forced lateral position, and even the rotary movements, have been observed in cases of demonstrated cerebellar lesion, but only when this

involved the processus vermiformis (Nothnagel).[1] According to Nothnagel, these symptoms have not yet been observed in connection with lesion of any other part of the brain ; not therefore with disease of the medulla. He admits, however, that they may occur in epileptic and hysterical conditions, and that the transitory assumption of a forced lateral position not unfrequently marks the onset of an epileptic attack.

Under these circumstances the phenomenon would be referred to medullary disease, though of a so-called functional character.

If now we suppose an irritation of the nucleus of the right spinal-accessory nerve as the starting-point of the morbid process, we may, from the clinical history, infer that this irritation has gradually extended upwards along the right half of the medulla. By the time it reached the nucleus of the acoustic nerve, a territory would have become involved whose lesion gives rise to complete rotations of the head and also of the trunk. Irritation of the floor of the 4th ventricle is moreover powerful in the production of nystagmus. This symptom, and that of increased oscillations, would then complicate the original affection, as we have seen that they did.

Further extension of the irritation would reach the upper extremity of the calamus scriptorius and the region lying between it and the corpora quadrigemina, which is considered to contain the principal vasomotor centre; hence the flushing of the face and

[1] The rolling of the head in acute hydrocephalus bears some resemblance to the rotary movements we are describing. But it might perhaps be possible to demonstrate that this symptom only occurred when, by distension of the aqueductus Sylvii, fluid had passed from the lateral to the fourth ventricle, and was exerting pressure on the medulla.

acceleration of the pulse which begin to coincide with the paroxysm. Whether the irritation was extended to the cerebellum, and whether during the paroxysm, hyperæmia really existed there and occasioned a rise of temperature perceptible at the occiput, we should perhaps hesitate to affirm, but it seems not improbable. There is only one symptom which could possibly indicate an original coöperation of the cerebellum, in the place of the secondary participation we have supposed. I mean the defective articulation. But it is difficult to be certain that this is really a morbid condition.

The observations on temperature were made previous to the publication of Dr. Amidon's essay, and on that account the lateral frontal region of the head was not examined in respect to temperature. It would certainly be most interesting to ascertain whether automatic muscular contraction occurring in muscles during a prolonged clonic convulsion, would be followed by the same alterations of cranial temperature as have been shown by Dr. Amidon, to follow voluntary contraction of the same muscles.

The crooning cry, which has been the latest addition to the symptomatology, must evidently be referred to clonic spasm of the inferior laryngeal nerve, derived from the spinal-accessory filaments associated with the pneumogastric.

In the absence of all other symptoms than those which have been described, and from the transitory and intermittent nature of the forced position and the rotary movements, we can hardly suppose an organic lesion to exist. We should rather infer a neurosis of an epileptiform nature, which, in its constant progress, is liable at any time to invade the pons, and occasion

an outbreak of true epileptic convulsions. Such an epileptiform character would explain the peculiarity of occurrence during sleep, period of repose for choreiform affections, and with them for the ordinary spasmus nutans.

I should be very happy if any member of the Society may pursue, further than I have been able to do, the analysis of this case.

V.

THE PROPHYLAXIS OF INSANITY.[1]

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A TERRIFIED popular imagination still pictures insanity as some mysterious and monstrous incubus, coming from distant regions of darkness to crush out human reason. In reality, however, insanity means a complex multitude of morbid states, varying indefinitely in form and intensity, but all composed of elements which preëxist in health. This fact affords a basis for prophylaxis, for it indicates the possibility of detecting these elements, and, to a certain extent, of anticipating their morbid combinations.

There are as many degrees in the soundness of men's minds as in the soundness of their digestions. Study of the organism of the family, sometimes in several generations, often serves to detect flaws in the individual organization otherwise too minute for notice. It is to the family organism that especially applies the doctrine of the blending of apparently opposite elements,—as genius and insanity,—both springing from an unstable equilibrium of the nervous system. These elements sometimes, though rarely, blend in the same person. But far more frequently it is inheritance from the undeveloped side of an organization of genius which results in an organization of imbecility.

[1] A portion of a paper read before the American Social Science Association, at Saratoga, Wednesday, September 7, 1881.

The original organization gives the physical substratum; upon this the succession of psychic processes, which begin with the dawn of consciousness, builds up the mental individuality. Ideas, feelings, volitions, enter liberally into the structure of the mind,—are the constituent elements of which this has been built up. Permit me to quote the description given by the celebrated Griesinger:

"Self-consciousness,—the Ego,"—he says, "is an abstraction in which are contained, closely welded together, a residue of all the sensibilities, thoughts, and volitions which the individual has ever experienced.

". . . These are gradually aggregated into complex masses of conceptions, varying in density and resistance, according to the internal cohesion of their elements. . . . The character of the individual varies with their relative predominance; their constant struggle with one another constitutes the internal conflict which is essential to normal mental existence.

". . . The development of insane delusions follows the same laws as that of healthy ideas. New sensibilities, volitions, and conceptions present themselves to the preëxisting conception-masses, are at first repelled by these, gradually penetrate them, and, if the cohesiveness of the latter be weak or weakened, assimilate to them until the Ego is transformed or completely falsified. *In this process the previous composition of the Ego is seen to be of immense importance.* A weak (loosely knit) nature will, much earlier than a strong one, be overborne by anomalous conceptions."[1]

Thus, at any given moment, the mental organism consists not only of its physical substratum, but of that *and* of the long series of psychic processes which

[1] "Pathologie und Therapie der Psychischen Krankheiten," 1867.

have been built upon it. It is a fundamental law of all organized tissues, and most conspicuously illustrated in the brain, that function not only depends upon structure, but ends by modifying it. Hence, morbid modifications of psychic processes may be initiated either in them or in the physical substratum. This is equivalent to the previous assertion, that insanity may be determined either by a psychic or a somatic cause, but generally requires the concurrence of both.

In the existing professional and popular reaction against the old puerilities of the exclusively moral theory of insanity, these facts are often overlooked or misunderstood. The question of prophylaxis has become narrowed down to the question of prophylaxis in marriage. This is not only much too narrow, and the social difficulties in the way very great, but the rules of practice have been by no means worked out, and many of those which have been suggested are erroneous or superficial.

The fact that the previous constitution of the mental conception-masses modifies the process of their falsification under the influence of mental disease, should suggest an effort to so build up this constitution that it may be fitted to resist strain. For the formation of the conception-masses is far from being a spontaneous or self-directed process. No ideas can enter the forming mind except from without, from communication with its fellows, or from the transformation of sense impressions. It is therefore largely in our power to determine the nature of the ideas of any child who is *thoroughly* guarded from his cradle. Again, the will develops in the mould it makes for itself by successive volitions; these may to a considerable extent be commanded or contrived. It follows

that, hand in hand with prophylactic treatment of the physical substratum of the inherited nervous organization, should go strenuous educational prophylaxis of the psychic processes. But there is needed a far-sighted, comprehensive, minute education, which should begin with the dawn of consciousness, and extend, if possible, through life. It should have a detailed objective or reason for each step in the elementary lesions of the disease which menaces the person, or in the elementary defects of his menaced constitution.

To assert that moral prophylaxis is useless because insanity is merely a symptom of physical disease, is to contradict the facts of the double nature and double origin of the psychoses which are admitted by the best authorities. Educational prophylaxis could only be expected to contribute one factor toward the solution of the problem; but it is one, and all the more worth considering, because at present it is so generally neglected.

A more plausible objection is, that the moral substratum of minds predisposed to insanity is peculiarly perverted, so that they are insusceptible of education. That it is precisely this insusceptibility which especially manifests their predisposition.

Finally, it may be alleged that the traits of character which exist in a person before an attack of insanity, can offer no guide for treatment, because in the attack these are all reversed.

This last objection is met by the answer that the prophylaxis of mental, as of somatic diseases, is to be directed, not to the symptoms of the malady, but to the constitutional defects which facilitate its invasion, and to the circumstances of the surrounding medium

which become the occasioning cause. Thus, it is known that under a great weight of responsibility, a cheerful-tempered, but feeble-willed person, may break down into melancholia. The prophylactic training should therefore be directed, not toward making such a person cheerful, but toward inuring him, by gradual practice, to bear responsibility. And so for other analogous cases.

The ideal prophylaxis implies that in neuropathic families the entire life of each child, its physical and moral training, and every detail of its social surroundings, should be planned with a view to avert mental disease. According to the degree of predisposition, this is liable to occur spontaneously at ordinary physiological crises, as puberty, menstruation, pregnancy, parturition, lactation, the climacteric ; or only under the influence of external causes. In the latter case, the far-sighted disposition of the social medium of a predisposed person may often avert an attack of insanity by averting the cause.

It is evident that the far-sighted and self-controlled guardianship required should be entrusted to a person not sharing the family constitution ; to the parent who may be exempt, or, if both are affected, to a person who is not a relative at all. For the present purpose, only a word is needed in regard to the main details of physical prophylaxis.

They are : abundance of nitrogenous food ; daily cold bathing ; pure air ; daily exercise in it, especially by means of cultivation of the ground, the cardinal employment of the body and mind of neurotics.

A fifth point of great importance is rest ; equally so for an immediately threatened attack, and in the life-long management of susceptible persons. For

them over-exhaustion and fatigue are always to be dreaded, and to these they are particularly prone, from the extremely deficient power of resistance of their nervous system. It is worth noticing that it is neuropathic families more than any others who are liable to neglect the foregoing precautions.

For effective moral prophylaxis, it is desirable that a certain amount of information be popularly diffused, to facilitate the awakening of domestic solicitude, the recognition of incipient insanity, and of the slighter but significant marks of the insane temperament. This may prove as useful as it has already done in regard to scrofula, rhachitis, tuberculosis, and other constitutional diseases.

Krafft-Ebing ranks severe and congenital hysteria with the psychic degenerations, and shows it to be the forerunner of much real insanity.[1] Knowledge of this fact might do much to check the capricious and vacillating treatment to which youthful hysterical patients are generally subjected. On the other hand, in the permanent prophylaxis for adult life, which must so largely be committed to the patient, it is extremely useful to be aware of the relative benignity of the very forms of insanity which usually excite the most alarm. Acute melancholia, mania, and primary dementia are classed with the functional disorders of psycho-neuroses, tending, under favorable circumstances, to spontaneous recovery. This knowledge might help to avert at least those distressing suicides which are committed, not from insane impulses, but under the dread of impending insanity. They are far from proving that this has already set in, for it is really not irra-

[1] This statement is not made in regard to acquired hysteria, symptomatic of uterine or other diseases.

tional to choose death in preference to permanent dementia.

The following traits are signalized as characteristic of the neuropathic constitution—constitution which affords the main physical and moral basis for the development of insanity.

In neuropathic families the children early manifest a remarkable nervous excitability, with tendency to severe neurotic disorders at physiological crises, as convulsions during dentition, neuralgias at menstruation. The establishment of menstruation is often premature, often preceded and followed by profound chloro-anæmia. The cerebral functions are easily disturbed, slight physical disorders being attended by somnolence, delirium, hallucinations. The nervous system seems to be everywhere hyperæsthetic. Reaction to either pleasing or displeasing impressions is excessive; there are abundant reflex neuralgias, vaso-motor irritations. Pallor, blushing, palpitations, præcordial anxiety, are caused by trifling moral excitement, or by agents lowering the tone of the vaso-motor nerves, as heat or alcohol.

The sexual instincts are precocious and often perverted. The establishment of puberty is often the sign for the development of spinal irritation, hysteria, or epilepsy.

The psychic characteristics correspond. The disposition is strikingly irritable and touchy; psychic pain arises for trifling cause; at the least occasion the most vivid emotions are excited. The subjects of this temperament alternate rapidly from one extreme to the other; their sympathies and antipathies are alike intense; their entire life is passed between periods of exaltation and depression, leaving scarcely any room for healthy indifference.

On the other hand, there is a remarkable inexcitability of ethical feeling. Vanity, egotism, and a jealous suspiciousness are common, and the temper is often violent. The mind is often obviously feeble, with few and monotonous ideas, and sluggish association of them. At other times ideas are readily excited, the imagination is active, even to the production of hallucinations; but mental activity is ineffective because of the rapidity with which it leads to exhaustion. There is no time to complete any thing before the energies flag. The will is equally deceptive in its apparent exuberance and real futility. Its capricious energy and innate weakness is a fit counterpart for the one-sided talent or even whimsical genius which often marks the intelligence.[1]

This disposition constitutes the moral substratum which, together with the physical constitution, affords the constitutional basis for psychic disease. In it two elements are conspicuous: a profound and often unconscious egotism, resulting from the predominance of the instincts over the faculties for external relations; and a constant ineffectiveness in the maintenance of these latter relations,—in other words, abnormal weakness of the will. These elements reappear in insane diseases. Egotism is the nucleus of the exactions of hysteria; and also determines the form of all delusions, which, whether primary, or engendered from emotional insanity, invariably centre on the depression or exaltation of self. The suspiciousness and violent temper so frequent in the neuropathic, develops easily into the technical delirium of persecution or of quarrelsomeness. The psychic hyperæsthesia common to several psychoses, but typical of melancholia, depends, on the one hand, on the

[1] Abridged from Krafft-Ebing.

same primitive egotism; on the other hand, on the weakness of the will, on account of which the normal channel from feeling to action is blocked. Pent-up feeling is always hyperæsthetic; psychic pain is the correlative of external ineffectiveness, even when not directly caused by it.

Diminished interest in external relations results in psychic anæsthesia, especially in regard to moral appreciations. This anæsthesia is again the direct correlative of the excess of instinctive and personal interests, and of the weakness of the will, which fails to enlarge the scope of the personality, as it is naturally destined to do.

When the will is feeble, sluggish, inert, the tendency of the mind to sink under pressure, and especially under the weight of responsibility, is very great. "The fact of human freedom," says Griesinger, "is the fact of the conflict in consciousness of opposing ideas, and of the termination of the strife by the conception-mass representing the Ego, which assimilates part of the ideas, and represses the rest." Feeble natures cannot bear this conflict without excessive pain, to which, at last, they not unfrequently succumb. In melancholia, the consciousness of diminished will power is a prominent and most painful symptom of the morbid state.

The feebleness of the will may be manifested, not by sluggishness, but by infinite caprice and incessant vacillations. This may reflect a torrent of incoherent ideas; or it may represent so rapid a transformation of an idea into an impulse that the latter alone seems to exist. Here the channel from the internal to the external world is not obstructed; its resistance, on the contrary, is abnormally diminished; yet the volition is

still ineffective. Effective volitions demand distinct and correct ideas of the external medium upon which they are to be expended. But one of the most essential elements of insanity, and of the constitution predisposing to it, is the diminution in the number, force, variety, and accuracy of the ideas held concerning the external world, and on the relations of the individual to it. This monotony of ideas is sometimes, before the attack, concealed behind desultory verbiage. Sometimes, during the immediate prodromata of an attack, it is temporarily replaced, even in feeble-minded people, by an unwonted vivacity and power. Completed delirium, however, is always monotonous. Correlated to the egotistic instinct, it always centres on the personality of the individual, which is outrageously oppressed, or illimitably exalted. The ideas are few; their associations sluggish; memory and attention are weakened even to extinction.

A deficient power of attention is generally a marked characteristic of the neuropathic state; it lies at the basis of the irritable impatience, which is so frequent in it. This leads to the formation of loosely knit conception-masses, ready to assimilate anomalous notions. The mind is naturally credulous; unapt for criticism. It offers less resistance than another to the invasion of false ideas.

Thus the three great elements in the moral substratum of a person predisposed to insanity, are: the egotistical predominance of the instincts over the faculties of reflection and external relation; the ineffectiveness of the will, even when this is impulsive or violent; the inaptitude for ideas, resulting in their poverty and imperfect combination. The whole nature is shrunken upon itself; there is not enough vital turgescence to

expand it to its normal circumference and to the points of contact of this with the external world.

The cardinal point in the management of such natures is, therefore, the expansion of their shrunken individuality. This is to be effected by means of a strenuous educational system, directed at once toward the repression of the egotistic instincts, the enrichment and systematization of the ideas, and, through multiplication of acts and external relations, the energizing of the feeble will.

The scope of the method will be made clearer by some examples. Thus: grief is an efficient moral cause of insanity. That it does not more often render people insane, is indeed a remarkable proof of the resources of the healthy human organism. However various the occasions for grief, yet in so far as these all imply personal loss, the principle of their influence is always the same.

The mind becomes so concentrated on the thought of this loss, that the latter acquires the ascendancy of a fixed idea. Apart from physical disease, the inability of diversion is great, in proportion to the habitual poverty and monotony of ideas; to the fewness of relations with the external world; to the preponderance of habitual interest in matters relating to self; to the inertness of the will, unable by vigorous action to expend externally irritations of psychic pain.

Similarly, when disappointment or humiliations, great or small, real or fancied, are the cause, or injuries, or the suspicion of injuries, the power of the predisposition and of the occasioning cause being constantly in inverse relation to each other, we reach a grade of exaggerated hysteria or hypochondria, where the egotistic instincts become able of themselves to generate melancholy, irritability, and delusions.

In another class of causations, shock plays a prominent part. Inability to resist shock is partly proportioned to poverty of ideas, which permit overwhelming surprises; partly to habitually unrestrained emotionality; partly to the passivity which prevents quick reaction. Analogous is the effect of strain, of excessive anxiety, of long-standing care and responsibilities. Healthy and justly proportioned indifference is essential to healthy equilibrium; an excess of sensibility over reflection or will power, predisposes to insanity under sufficient irritation. All experience shows that an excess of egotistic sensibility is far more dangerous than an excess of sympathy, the latter being indeed extremely rare in the neuropathic constitution. It may become a cause in non-constitutional insanity. Another line of causation is that in the direction of ideas, where the invasion of false ideas is facilitated by habits of credulity, superficial reasoning, loosely knit conception-masses. An unreflecting enthusiasm easily embraces exciting doctrines, as in the various religious or political manias, or is carried away by suggestions which covertly appeal to the egotistic instincts, flattering or alarming them, or submits to incongruous beliefs, as in the so-called partial insanity or monomania.

Perhaps none of the details of an educational prophylaxis are foreign to the principles theoretically advocated for ordinary education. But in this they are applied, if at all, in a manner so lukewarm and vague as would render them useless for so grave a problem as the prophylaxis of insanity. To consider these principles in the order already enumerated: the repression of egotistic instincts demands effort in two directions. Negatively, these are to be atrophied by

a studied atmosphere of indifference to caprice, violent tempers, ridiculous pretensions, exorbitant exactions; none of which are allowed to be gratified. In this permanent atmosphere, created by the mind controlling and guarding the child, he may learn to appreciate his insignificance relatively to the external world. Toward this and its interests he is secretly apathetic, except so far as they may be made subservient to his own vanity. The principle of justice, based on the simple fact of primitive equalities, must be profoundly in-wrought, by practical exercises, into the consciousness of the neurotic. He is naturally inclined to submit every thing to the test of his sympathies and antipathies; and the cultivated habit of reference to simple justice instead, will save him from innumerable entanglements, perplexities, and agitations, most dangerous to his mental equilibrium.

The multiplicity of human interests, the vastness and importance of the interests of the world, as compared with his own, may be impressed upon the child's imagination in many ways, if ingenuity be not lacking. The incidents, utilized or contrived, necessarily vary with the age of the child, but the same complex end is always to be held in view: restoration of the normal proportion between egotistic instincts and faculties of relation, and excitation of healthful ideas through healthful practical experiences and association with the fortunes of his fellows. Sometimes together with mental vivacity, sometimes with mental inertness, the mind of the neuropathic individual is apt to be really indifferent to intellectual relations, to knowledge for its own sake, to disinterested curiosity, the happiest appanage of a sound intelligence. Interested motives must be skilfully supplied, sufficiently to provide for

the acquisition of knowledge essential to the enrichment of ideas, yet with caution, lest vanity and *amour propre* be unduly stimulated.

The acquisition of knowledge, the training in morals, the formation of habits of thought, must all be centred upon practical activities. It is the proper development of these which is to be relied upon to energize the feeble will; to accustom it to effectiveness by training to productive industry; to broaden and deepen the channels from internal concepts to impulses; to provide thus for the overflow of dangerous irritations; to check the flightiness, frequent forerunner of insane impulse; to widen the range of interests and of correlative ideas, and hence of resource against shock, vexation, and misfortune; to moderate inordinate vanity by submitting its pretensions to practical tests; to regulate moods by habits of daily labor; and to enlarge the entire personality, for the future as well as the present, by insuring, from internal pressure, the creation of a permanent career. This latter element of prophylaxis might well save from insanity many of the "lazy and languishing young ladies" whom Mortimer Granville complains of as filling private insane asylums.

It is not enough to attempt to widen the range of ideas. In some directions, and unguarded, this proves simply disastrous to persons of innately feeble intelligence. They must be trained in the formation of practical concepts; associated as much as possible with practical facts, with sense impressions, and with experiences in action. Clearness, definiteness of ideas, their frequent association with images, afford no inconsiderable safeguard against morbid mental confusion. Similarly the careful training of the senses in

various techniques contributes much toward the steady outward direction of nervous energies, which is needed to counteract the tendencies to internal concentration.

In this connection gymnastic training has a mental as well as a physical influence. It would be difficult to prove that such training of the periphery of the nervous system could counteract the development of hallucinations, which are caused by central irritation of the sensory centres. But it certainly lies in the line of such counteraction.

If it be important to fill the mind with concrete ideas, it is at least as important that these be correct, and not liable to be uprooted in later life. This liability constitutes a real danger in the notions of popular theology, which are so loosely allowed to be acquired even by guardians who do not believe in them. To persons predisposed to insanity, the uprooting of fundamental ideas can by no means be performed with impunity. It is important to train such persons early in a sound and simple philosophy, which shall provide a firm basis for thought and life without inviting to speculative thinking.

Finally, since the object to be gained is firmness and strength for the mind in dealing with its own concepts, practical exercises in the elementary intellectual acts are extremely important. These are but feebly carried out in ordinary schools, because the object in view is not distinctly perceived or firmly grasped. The first signs of failing mental power are : loss of memory, of power of association of ideas, of summoning contrasting ideas into consciousness, of reproducing or comparing or criticising them. It is indicated, therefore, to train the mind in advance to profound habituation with these various processes. Such train-

ing it is true will avail nothing when physical lesions have begun to destroy the intellectual mechanisms. But it may avail much in the cases where the integrity of these first becomes impaired from obstruction of function and psychic disability.

One other detail deserves notice, for it rarely receives attention. In minds predisposed to insanity there is often, perhaps always, a marked deficiency of elasticity. An impression sinks and remains ; the mind cannot disengage itself nor recover its tone ; it cannot pass quickly enough into the contrasting mood. Now the capacity to do this is the natural provision against strain : it probably corresponds to a law of rhythmic action in the physical mechanisms of thought. This capacity should, therefore, be carefully cultivated by encouraging alternations of attention at the first sign of fatigue. The contrary practice of forcing an immature mind to continued attention while under the influence of fatigue, instead of teaching it how to quickly change, is the habit of commonplace education. Injurious to all, it is especially so to persons predisposed to depressing forms of insanity. It exhausts still further the elasticity in which they are naturally deficient.

The management of the perverted instincts of neuropathic constitutions may, when these are advanced in deterioration, prove a hopeless task. At a less severe degree, however, many bad propensities may be held in check by a skilful combination of the methods of punishment,—emulation and distracted attention.

One difficulty in guiding these cases generally lies in the fact that their pathological nature is not early recognized. Children are incessantly moralized, whose minds do not contain any conceptions of mor-

als, and only an imperfect mechanism for ethical functions. According to the degree of imperfection, such persons must be dealt with as animals, who can certainly be trained into habitual lines of conduct, even though destitute of the corresponding abstract ideas.

One morbid appetite calls for special mention, that, namely, for alcoholic liquors. This, like the others, is often manifested early in life, and, as known, is not only a symptom of a neuropathic constitution, but, when indulged, a potent occasional cause of insanity. The management of this appetite is a most difficult problem. It has been plausibly suggested that the permanent and moderate administration of alcohol in the form of beer, might, with other treatment, help to avert the development of the irresistible craving.

Such are the abstract principles of a system of treatment, which, if seriously carried out, properly associated with physical treatment, and so arranged that every other consideration should be subordinated to the attainment of its ends, should prove of real value in helping to avert many cases of insanity.

VI.

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"ANTAGONISM BETWEEN MEDICINES AND BETWEEN REMEDIES AND DISEASES."—Cartwright Lectures for Year 1880. By ROBERTS BARTHOLOW, M.D., Professor of Materia Medica and General Therapeutics in the Jefferson Medical College of Philadelphia, etc., etc. D. Appleton & Co. 1881. pp. 122.

The Cartwright lectures have been inaugurated most auspiciously by Dr. Bartholow. He has compressed into a narrow space a brilliant summary of the facts at present known in regard to one of the most fascinating questions of modern medicine. The demonstration of a precise antagonism between the action of drugs, has a double bearing on the theory of therapeutics. On the one hand, new practical resources are placed at our disposition, not merely to meet the accidents of poisoning, but, as we may hope, to combat symptoms similar to such accidents, when they have arisen spontaneously in the course of disease. But a further and a more purely philosophical interest attaches to the study of the toxic symptoms, for the reason that their exact (remote) cause is known, and known to be an agent within our grasp.

The very existence of such a definite train of symptoms proves that we are able by external agencies to modify, in a given direction, the processes of a living

organism. This fact is in formal opposition to the fundamental doctrine of Medical Nihilism, which says: "It is absurd to attempt to modify anatomical conditions by means of drugs." In view of the palpable contradictions to this doctrine which the facts of toxicology afford, one of two conclusions must be admitted. Either the symptoms induced by poisons are independent of anatomical conditions; or else by the administration of a drug, we *are* able to change the anatomical conditions of health to those characteristic of an artificial disease. It is true that the condition thus voluntarily induced are only similar to those of natural disease, and by no means identical with them. "We can," observes one of the most eminent authorities on artificial pathology, "imitate symptoms but not diseases. We can render an animal diabetic or epileptic, but we cannot create diabetes or epilepsy."[1] Nevertheless, this imitation is already of the greatest importance. And when, in studying the effects of one poison we find that they can be combated by the appropriate use of another, and that this second poison can be shown to be capable of initiating a train of symptoms exactly the opposite in appearance to those which have been caused by the first, a horizon certainly opens before us of a rational therapeutics, destined to encroach more and more on the therapeutics of pure empiricism.

The hope of such a future is distinctly communicated by Dr. Bartholow, even in the title of his lectures. Consideration of the "antagonism between medicines" is immediately followed by discussions on an analogous antagonism "between remedies and

[1] Vulpian. Leçons sur les maladies de la moelle épinière.

diseases," and to this latter subject are devoted two out of the six lectures of the course.

It is on the "scientific application of the principle of antagonism to medical practice" that the author seems to rely, to reverse the severe judgment pronounced on materia medica by Bichat, in 1818. "It is a collection of incoherent opinions,—is, perhaps, of all the physiological sciences, that which most exhibits the contradictions of the human mind. In fact, it is not a science for a trained intellect; it is a shapeless mass of inexact ideas, of observations often puerile, of imaginary remedies strangely conceived and fantastically arranged. It is said that the practice of medicine is repulsive. I go further than this: it is, in respect to its principles taken from our materia medicas, impracticable for a sensible man." (Quoted, p. 13.)

Piquant indeed is the contrast between the uncertainty thus pungently described, and the exquisite precisions which, according to our author, may even now be predicted of so many therapeutic manœuvres. We would not deny Prof. Bartholow's energetic optimism. Optimism, even when exaggerated, often serves, like the flag of the color sergeant, to lead a substantial advance. But in estimating the resources at our disposal for the removal of disease, we think it is of great practical importance to bear in mind the (often unknown) *tertium quid*, which distinguishes morbid processes of spontaneous, *i. e.*, internal origin, from those which have originated in external influences, whether traumatic or toxic. The problem for somatic diseases is the same as for insanity: health failure at any one point of the organism very often, if not always, implies deviation of the entire organism from the norm. It is this general health failure, as we

are inclined to believe, which is at least one cause of the frequent failure to allay spontaneous symptoms by remedies which have been successfully antagonistic to the same symptoms when artificially induced.

Did we follow Dr. Bartholow literally, we might infer that the different success in the two cases really depended on an absence of anatomical lesion as a basis for toxic symptoms. We are told to select our therapeutical agents on the basis of "physiological antagonism." And this "means simply a balance of opposed actions on the same tissue. It does not induce a change of structure. The opposing agents counterbalancing each other, the functional disturbance subsides, and the normal equilibrium is restored." (P. 11.)

But physiological actions are inconceivable except as the concomitant of the molecular changes in the elements in function. The difference between each molecular change and gross palpable lesions of structure, is one of degree not of kind. An agent that causes arterial tension by relaxing the peripheric arterioles, determines a rearrangement of the molecules in their muscular coat. An antagonistic drug which should raise the tension by really acting on the same arterioles, must necessarily reverse the molecular arrangement effected by the first. The objective of the second drug is not the "opposing action of the first," but the tissue which has been modified by that.

But there are further objections to Dr. Bartholow's formula. We think it can be shown, even from his own summary of facts, that "opposed actions on the same tissue" never take place except in one direction. When a tissue or organ is paralyzed by any poison, it fails to respond to other poisons which ordinarily have a tendency to stimulate it. This failure is ob-

served whether the paralyzing agent be administered first, or when the stimulating agent is in full operation. In the latter case, the stimulating poison is effectually antagonized. It is on this account that, as Dr. Bartholow himself remarks, the list of antagonisms effected by atropine is so large: it paralyzes so many "end-organs." Paralyzing the ciliary branches of the third nerve to the pupillary sphincter and to the ciliary muscle, atropine antagonizes all drugs which cause myosis, either by stimulating the third nerve, or by antagonizing the ciliary muscle or circular fibres of the iris.[1] Thus, it antagonizes pilocarpine, eserine, muscarine, and the initial action of morphine. In the later stages of morphine poisoning, where vaso-motor paralysis of the iridian blood-vessels increases the myosis by turgescence of the iris, the counteracting effect is aided by its influence on the circulation. Now, in all the above cases, the antagonism of atropine to the myotic drugs is not reversed. When the pupil has been dilated by atropine, it is admittedly difficult to counteract it by any antagonist. In the most famous and thoroughly discussed antagonism, that between morphine and atropine, Dr. Bartholow declares that the pupil offers no sure guide, and that the action of atropine preponderates. Muscarine will not contract the pupil dilated by atropine. (See p. 63 of Lectures.)

According to Bartholow the "atropinized pupil resists the action of eserine." (P. 54.) If, however, as Galezowski declares, eserine discs will contract a pupil

[1] Dr. Bartholow admits, in several places, that atropine "stimulates the radiating fibres of the iris"; but of this we know of no proof. The experiments upon the excised eye, we believe first performed by Brown-Séquard, only demonstrate that atropine acts on nerve terminations, and that the central communication of the third nerve is not essential. This is precisely analogous to its action on the terminal branches of the vagus, after section of the trunk.

so dilated, it would be by directly tetanizing the circular fibres of the iris ; thus there would be no "opposed action" on the third nerve.

Quite similar observations hold true of the heart. Here again the "antagonism" of atropine is extensive and conspicuous, because it paralyzes the terminal fibres of the vagus in the cardio-inhibitory ganglion. Thus it antagonizes, in Dr. Bartholow's sense, by "opposed action on the same tissue," all the drugs which slacken the pulse by stimulating either the central or peripheral portion of the inhibitory apparatus. Thus, it is antagonistic to digitalis, to morphine in its early stages, to muscarine. But the experiment is classical in toxic experimentation, wherein the heart, arrested by muscarine, may be set to beating by atropine, while the atropinized heart altogether refuses to respond to muscarine. When morphine succeeds in reducing the pulse accelerated by atropine (and this is admittedly difficult), it does so by diminishing the excitability of the excito-motor ganglia. Here again, therefore, there is not "an opposed action on the same tissue," but a similar, *i. e.*, paralyzing action on a very different tissue.

Similarly, atropine will arrest the salivation caused by physostigma or pilocarpine, for it paralyzes the chorda tympani. When this paralysis has once been effected, salivation is no longer possible. Chloral will moderate the convulsions caused by strychnine ; there is no proof that strychnine will avert the respiratory paralysis threatened by toxic doses of chloral.

Dr. Bartholow admits this last fact with great surprise. We consider it rather as an illustration of a general law that we have already indicated, and which may be thus formulated :

"The response of an organ to a physiological or toxic stimulus, may be prevented by paralyzing the organ. But paralysis of an organ cannot be antagonized by stimuli addressed to the organ, since the paralysis implies that susceptibility to impressions has been lost. Cure of paralysis can only be obtained by elimination of the paralyzing effect. During the process of elimination, the effects of the paralysis may often be combated by stimulation of other organs remaining able to respond. This constitutes a net antagonism to the effects of the poison, often effectual, but always indirect."

It is this form of antagonism which is to be inferred from the "physiological basis" described by Dr. Bartholow. Part of this basis is afforded by the mechanisms which exist throughout the body for systemic alteration of functions, with consequent "restraint of activities within proper limits."

"If there were not some antagonism to the spasm centre, every trifling peripheral irritation would produce most extravagant reflex effects. . . . The movements of the vessels are regulated by a vaso-motor centre in the medulla. By the opposed action of the dilator and constrictor forces, the vascular tonus is maintained at the normal. A similar mechanism controls the cardiac movements; there is a motor apparatus for carrying on the action of the heart, and a regulator apparatus for restraining the movements within proper limits. . . . If the arterioles suddenly dilate, the blood pressure as quickly falls, but danger to the circulation is prevented by an increased action of the heart. . . . Here opposing forces maintain their equilibrium." (P. 21.)

The presumption is that artificial antagonism to a

given process in an organ will be best effected by acting upon the apparatus which provides for physiological antagonism to the same process.

If we apply this principle to some of Dr. Bartholow's favorite illustrations of antagonism, we shall discover quite a different interpretation of them from that given in these lectures. For instance, atropine is said to "stimulate respiration," because accelerated respiration is a phenomenon induced by atropine. Hence atropine is considered a valuable antagonist to any poison threatening death by "respiratory paralysis."

Now, it must be observed, in the first place, that each of these opposed terms is not simple, but extremely complex. The acceleration of the respiration may depend upon several circumstances, and so also its slackening; and special inquiry is necessary before we can be assured in any given case, that these are exactly opposed to each other. Analogy, at least, would suggest that atropine paralyzes the inhibiting respiratory centres,[1] and that the respiratory movements are thus accelerated in the same way as the cardiac, when their inhibitory apparatus is paralyzed. In antagonizing morphine, the same succession of events presents itself for the respiration as for the heart at the beginning of morphine poisoning. The respiration may be slowed, because the increased intracranial pressure has stimulated the inhibitory centre of inspiration, as it has the roots of the vagus and of the motor oculi nerve. Then the paralyzing effect of atropine would be beneficially antagonistic. Later on, when the susceptibility of the inspiratory centre itself is becoming benumbed, it might be (according to our theory) indi-

[1] Described by Rosenthal, Bemerk. üb. d. Thätigkeit d. automatischen Nervencentren, etc. Erlangen, 1875.

rectly aroused by more rapid capillary circulation both throughout the tissues and in the medulla itself. By accelerating the circulation, therefore, atropine brings to bear upon the inspiratory centre the normal blood-stimulus to which it is physiologically adapted to respond. The antagonism to the effect of the morphine would therefore be indirect.

We would note, in passing, that the common assertion (which Dr. Bartholow endorses), that morphine induces carbonic acid narcosis, seems to us very inaccurate. The characteristic reaction of the inspiratory centre to an excess of carbonic acid in the blood is convulsion, which morphine does not cause in adults. We think it could be shown that the slackening of the respiratory movements coincides with, and follows, diminution of molecular respiration in the tissues. The phenomena are those of apnœa, not of asphyxia ; there is not an excess of carbonic acid irritating the inspiratory centre, but a deficiency, and leaving it in abnormally long intervals of repose. Hence, might be suspected another mode of action of atropine, in antagonizing morphine, viz., an acceleration of the circulation and tissue-change. But into speculations like these, Dr. Bartholow does not enter. His summary, however, contains many illustrations of the doctrine we maintain, namely, that effective antagonism is always either paralytic or indirect. Thus, having no direct control over the cardiac tetanus of angina pectoris, we can yet relieve the attack by paralyzing the contracted arteries through inhalations of amyl nitrite. Failing to arrest uterine hemorrhage by astringents directly applied to the bleeding surface, we may effect our purpose with nux vomica, which "stimulates the cardiac and respiratory centres."

And so on. The more examples we multiply, the less should we be ready to accept Dr. Bartholow's mutual antagonism by means of "opposed actions in the same tissues"; the more inclined to believe that the antagonistic influence is necessarily exerted upon different organs, or upon tissues in the same apparatus.

We have selected for comment the topic that happened to attract our attention. We leave to others the agreeable task of seeking food for other reflections from these most suggestive lectures.

VII.

HYSTERICAL LOCOMOTOR ATAXIA.

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ELLEN R., an Irishwoman, aged thirty-five years, consulted me first in the spring of 1878. She was a widow, and had had one child fifteen years previous to the time of my seeing her. She stated that her father had been insane for twenty-five years, and during the time that the patient was under observation, a brother became also insane, and remained so.

The patient, a cook by profession, was a woman of sufficiently robust build, who claimed to have been in good health, not only before, but for several years after, her confinement. During seven years, however, her health had been impaired.

The first symptoms of ill-health pointed to uterine disturbance: leucorrhœa, to which the patient paid little attention; and vesical tenesmus and pain at micturition, which often were quite distressing. Not until several years later did she experience any pain about the pelvic region; and then it was moderate and inconstant. More frequently she suffered from pains in the thighs, and more especially from numbness in the same region. Menstruation was regular and painless, but too profuse.

Nervous symptoms, however, of manifold character, soon appeared, and masked those of local significance.

The first onset of these consisted in an attack, which she described as a "fright," coming on as she was about to take some medicine for the vesical tenesmus. Apparently there must have been a spasm of the œsophagus, for she fancied she was about to have hydrophobia. It was accompanied by vertigo, diffused numbness and prickling throughout the body, and a feeling as though she would fall forward. These symptoms passed away after a few hours, but the patient remained so weak that she was unable to walk for several months. During this period of idleness she was subject to fits of weeping without cause.

It was three years later, and several months before she consulted me, that the disturbances of motility, which henceforth became so prominent, began. The patient first experienced difficulty in going up and down stairs; then, in rising to her feet from a kneeling posture; finally, her gait in walking became extremely unsteady—she swayed from side to side as she progressed, and often tried to support herself by surrounding objects; the numbness in the limbs increased, but there was no pain.

It was for this difficulty of locomotion that the patient consulted me. On the first, and somewhat hurried, visit, I found that the uterus was slightly enlarged—the sound passing to a depth of eight centimetres—and considerably prolapsed. I inserted a cup-and-stem pessary, and showed the patient how to adjust it. This instrument effected so great an improvement in her power of locomotion and of rising from the ground, that she ceased to visit me, and further examination of her case was thus postponed.

The improvement lasted two or three months, but then ceased; the difficulty of walking returned and

increased; patient grew weak and was obliged to give up work. During the summer and fall of this year she entered successively three hospitals—the Homœopathic, St. Luke's, and the Presbyterian. In both the latter a positive diagnosis was made of locomotor ataxia,—diagnosis subsequently defended with earnestness in a personal conversation I had an opportunity of holding with one of the physicians who made it. This fact is mentioned as showing the deceptive character of the symptoms in question.

I next saw Ellen R. in Jan., 1879, after an interval of six months. I found the difficulty of locomotion so much increased that the patient was obliged to use a cane in walking. The gait was uncertain and staggering; the arm not employed with the cane, oscillating, the body being inclined forward. She would walk at first slowly, then hurriedly; the foot was thrust incoherently forward in different directions and then brought suddenly down to the ground. It was this element of the gait which seemed most distinctively ataxic. The uncertainty of gait was increased by closing the eyes, and the patient could then with difficulty maintain her equilibrium. In this position, however, she resisted efficiently, firm downward pressure upon the shoulders. Recumbent, all movements of the limbs could be executed; the ataxia disappeared. This was an important difference from the symptoms of tabes dorsalis.

At this date there was no disturbance of sensibility in the limbs—neither pain nor anæsthesia. There was, however, a sense of constriction in the abdomen, extending toward the epigastrium, but not around the back. There was also complete absence of the patellar tendon reflex. One sensitive point existed in the

cervical spine. Faradic contractility was intact. The patient was slightly deaf in the right ear, and complained of impairment of vision of right eye, where, indeed, the optic nerve was partly atrophied. This circumstance had been recognized in the hospitals, and considered confirmatory of the diagnosis of locomotor ataxia. But there had been a mechanical injury to the eye, and closer examination showed that this was the cause of the impairment of vision.

During the last six months the patient had had several attacks of dysphagia, such as had ushered in her entire illness, but much more severe. In addition, she had become subject to violent attacks of dyspnœa, with alarming sense of suffocation. The voice had become habitually affected; words were uttered spasmodically, the patient frequently catching her breath while she spoke, and usually terminating her sentence in a whisper.

The patient had abandoned the use of the pessary, because she had had pain from it. The uterus was somewhat prolapsed—though less so than formerly,—heavy, and congested. Much endocervicitis existed, and it was now observed that the cervix was lacerated.

The patient was taken into the New York Infirmary, the endocervicitis treated with carbolic acid and glycerine, and prolonged galvanic applications made to the spine. Under this treatment, with rest and good food, she improved greatly. By the middle of March she was able to walk a considerable distance. In April the pessary was reapplied, and faradization of the limbs was substituted for galvanization of the spine. In July the patient left the infirmary, and immediately began to feel worse.

I saw her again in September, 1879. The ataxic

gait had returned. In addition, the patient suffered from pains shooting across loins, then from hip to ankle, "as if it darted all through." The pains simulated the fulgurating pains of locomotor ataxia, but the "stabbing" pains, on whose diagnostic value Seguin has laid much stress, were absent. The patient had a cotton-wool feeling under the sole of the feet, principally the great toe. As before, the tendon reflex was entirely absent.

I still, however, persisted in the diagnosis I had made, of hysterical ataxia, primarily induced by uterine disease; and explained the relapse by the existence of the laceration of the cervix. This caused few or no symptoms while the patient was at rest, and while the endocervicitis was being treated; but became an efficient cause of irritation when the patient resumed work, walking, standing, etc.

Toward the end of October I operated on the cervix, with complete success; at the end of two months the uterus was normal in size and weight, kept in position; the cervix perfectly healthy.

At this time the patient "felt much steadier on her legs." The ataxia and shooting pains disappeared, though there was still some swaying of the body while the patient walked. Facility of going up and down stairs has greatly increased.

Throughout the winter, the patient having returned to service, her health remained only passably good. She was weak; suffered from pains in hips and legs; occasional cramps or patches of rigidity on inside of foot or outside of thigh. In March of 1880, patient began to suffer from twitching in left leg at night, and both limbs began to be markedly paretic. The paresis was manifest even in the recumbent position;

the limb was lifted or moved in a lateral direction, sluggishly and with a sense of great effort.

The patient always felt worse in the morning; at first could hardly move, but having been on foot some time, could stand or walk pretty well; always improved by application of faradic current to limbs. After a few minutes' application, much stronger contractions were obtained with the same strength of current.

As six months had now elapsed since the operation and the removal of all uterine lesion, I began, for the first time, to fear that after all some form of chronic sclerosis of the cord existed, more probably lateral than posterior. Several symptoms were, however, lacking, but the recent occurrence of "cramps" seemed to herald the development of the rigid contractures and of the tremors characteristic of lateral sclerosis.

At this point the patient ceased attendance. She continued to grow worse; could not get up and down stairs; had shooting pains all over her body; lost flesh and appetite. Finally, she went out West (in August, 1881), travelled a good deal, remaining in service in different places according as her health would allow; had several attacks of chills and fever, and of "congestion of the liver." But in the midst of these febrile disorders, and perhaps because of them (?), the hysterical symptoms gradually subsided. In September, 1882, she returned to New York and came to see me. I found her entirely free from all her former troubles, only a very slight swaying of the gait recalled the paresis and ataxia of former times; she could go up and down stairs, rise from her knees, etc., without difficulty.

Coincidently, the aphonia, dysphagia, and dyspnœa

had disappeared; the patient suffered from pains nowhere.

The tendon reflex, however was still absent.

The diagnosis in this case rested upon :

1st. The marked modification of the symptoms determined by each modification of the uterine disease: in the first place by the use of the pessary; in the second, by treatment of the endocervicitis; in the third, by the operation on the lacerated cervix.

2d. The fact that ataxia of the lower extremities preceded all modifications of the sensibility. In tabes dorsalis some degree of anæsthesia (Rosenthal) of stabbing, or of fulgurating, pains (Seguin), always precedes any marked ataxia. It is conceivable that an hysterical anæsthesia should have existed in the case of Ellen R., and thus increased the difficulties of diagnosis, but it did not.

3d. The coincidence of paresis of the lower extremities, with or indeed out of proportion to the ataxia, as shown by the difficulty of rising from the knees, of going up stairs, etc.

4th. The absence of ataxic incoherence in the movements executed in a recumbent position.

5th. The absence of pupillary phenomena, bilateral atrophy of the optic nerves, gastric attacks; the presence of paroxysmal dysphagia and dyspnœa, both predominantly hysteric phenomena; the limitation of constriction to the abdomen.

The diagnosis was, however, particularly obscured by :

1st. The existence at one time of pains a good deal resembling fulgurating pains.

2d. Of plantar anæsthesia, cotton-wool feeling under sole of foot. This, however, did not appear till late

in the disease, and on close examination, seemed to be limited to the great toe.

3d. The persistent absence of patellar tendon reflex.

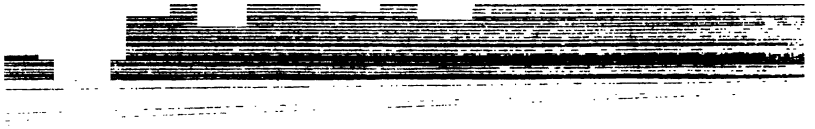
4th. The recurrence of symptoms after removal of all peripheric irritation in the uterine system.

5th. The appearance of localized cramps and tremors in different parts of the lower limbs. On the whole, however, the diagnosis could be and was made out; and was confirmed by the result. But its difficulty is best shown by the fact that the contrary diagnosis was made in two hospitals, by most competent physicians, who, however, only had the patient under observation during a short space of time.

The mechanism of the production of such symptoms, by means of an irritation, starting from the pelvis, is certainly very obscure. We must infer that the centripetal impressions arriving from the focus of irritation are distributed, on the one hand, to the posterior roots in the columns of Burdach; on the other hand, to the lateral columns of the cord, in such a way as to produce respectively ataxia and loss of tendon reflex, as sclerosis of these same parts would have done. Since the early affection of sensibility in tabes dorsalis has been associated with the debut of the process in the columns of Goll, the absence of any sensory disturbance in the early history of our case becomes all the more important in excluding this portion of the cord from even functional disease, and thus in distinguishing the case from true locomotor ataxia.

The loss of tendon reflex—instead of the exaggeration of it, so often seen in hysteria—implies an inhibition of the motor impulses transmitted through the lateral columns, — the inhibition dependent on irritation of posterior nerve-roots from pelvic irrita-

tions. The inhibition constitutes a functional imitation of the effect which may be elsewhere produced through destruction of these same columns in organic disease. The ataxia and paresis must be similarly explained; as also the attacks of muscular rigidity transiently observed. The discovery by Charcot of organic disease—sclerosis of the lateral columns—in a case of hysterical contraction persisting till death, indicates the extent of possible affection of this part of the cord in hysteria.



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